THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—22ND YEAR. SYDNEY, SATURDAY, JULY 27, 1935. No. 4.

Table of Contents

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ORIGINAL ARTICLES— PAGE.

"The Investigation and Treatment of Vaginal Discharges", by HUBERT JACOBS, V.D., M.D., F.R.C.S., F.R.A.C.S., D.G.O. .. 101

"Sedatives in Acute Cerebral Trauma", by GILBERT PHILLIPS, M.Sc., M.B., B.S. .. 108

"The Practical Method of Dealing with Asthma and Hay Fever", by ROBERT S. STEEL, M.B., Ch.M., M.R.C.P. .. 114

REPORTS OF CASES—

"Schilder's Disease", by A. HOLMES À COURT, M.D., F.R.C.P., and OLIVER LATHAM, M.B., Ch.M. .. 117

"Diffuse 'Encephalitis', Presumably Schilder's Disease", by JAMES FLYNN, M.B., Ch.M., D.O., D.O.M.S., F.R.A.C.S., and T. M. GREENAWAY, M.B., Ch.M., M.R.C.P. .. 120

LEADING ARTICLES—

Infant Mortality and Illegitimacy .. 121

CURRENT COMMENT—

The Treatment of Spasmodic Dysphagia .. 122

Psoriasis .. 123

ABSTRACTS FROM CURRENT MEDICAL LITERATURE— PAGE.

Physiology .. 124

Biological Chemistry .. 125

BRITISH MEDICAL ASSOCIATION NEWS—

Scientific .. 126

Annual Meeting, Melbourne, 1935 .. 127

CORRESPONDENCE—

Vaccination against Smallpox .. 130

The "Chest" Physician and the Radiologist .. 131

The Mortality of Appendicitis .. 131

Sympathectomy in Retinitis Pigmentosa .. 131

Chronic Infective Arthritis .. 131

CORRIGENDUM .. 131

BOOKS RECEIVED .. 131

DIARY FOR THE MONTH .. 132

MEDICAL APPOINTMENTS .. 132

MEDICAL APPOINTMENTS VACANT, ETC. .. 132

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 132

EDITORIAL NOTICES .. 132

THE INVESTIGATION AND TREATMENT OF VAGINAL DISCHARGES.[1]

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THE investigation, management and treatment of vaginal discharges is a subject of common interest to both gynæcologist and general practitioner, and one which presents many difficulties. Prompted by the frequency with which such cases are referred to me, I thought that a paper dealing with this symptom would not be without interest. At the outset I should like to say that no claims are advanced for originality—rather I hope to be able to present to you a comprehensive survey of what is known today concerning vaginal discharges. Although they are so frequently met with in general practice, yet many difficulties are encountered when investigation is commenced, and very often an exact diagnosis has not been arrived at before imperfect or inadequate treatment is commenced. Such a procedure merits lack of success, by no means infrequent, and the patient shows the obvious signs of disappointment and dissatisfaction.

The scope of this paper includes not only discharges of the vagina, but also those originating from neighbouring foci. The history of medicine is rich with references from the Greeks and the Latins to the condition of the white flux of women, and this was still described in the sixteenth century writings of Ambroise Paré. In more recent times the word leucorrhœa is used to describe this condition, derived from the Greek λευκός, white, and ῥοία, a flow or flux.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on June 5, 1935.

Strictly speaking, therefore, the term should apply only to an excess of the white discharge, the physical, chemical and bacteriological characteristics of which are within the limits of normality. Nowadays, however, leucorrhœa is broadly applied to describe all discharges from the vagina other than actual hæmorrhage, and for the purposes of this paper I shall define leucorrhœa as that amount of discharge which is greater than the usual, which necessitates the wearing of a diaper, and which may or may not be coloured, irritating or odorous. Thus such a discharge may be serous, mucous, purulent, and at times sanious. In addition, urinary and fæcal vaginal discharges must be included.

The Normal Vaginal Secretion.

The ætiology of vaginal discharges will be more fully understood if I discuss the origin as well as the physical, chemical and bacteriological characteristics of the normal vaginal secretion of the healthy adult. In order to cover this ground I must briefly refer to some considerations of the embryology, anatomy and physiology of the female organs and of the influence of the endocrines on their development. It is important to realize that great changes occur in the female genital organs during different epochs of her life, such being both histological and anatomical. To day we are in a position to state conclusively that such changes are controlled by the activities of her endocrine system, and particularly the hormones of the anterior pituitary and the ovary. These epochs or phases are four in number and are: (i) at birth and the latter months of intrauterine life, (ii) from infancy to puberty, (iii) from puberty to menopause—the reproductive period of life, (iv) post-menopausal.

A consideration of the vagina and its secretion at each of these phases is necessary here, for such details are not generally appreciated and the knowledge greatly simplifies the explanation of the process of production of many forms of discharge. The genital organs of the normal new-born female infant are not fully developed either anatomically or histologically. The vagina at birth is sterile and remains so from twelve to twenty-four hours. There is secretion present at this time, often copious, creamy white in colour, non-mucoid, and acid in reaction, but within three to four days Döderlein's *Bacillus vaginæ* is found to be abundant and in a pure culture. At the end of the first month a marked change occurs in the secretion, which becomes scanty, mucous, and much less acid, being often neutral or faintly alkaline. Bacteriologically, many other organisms are now present — cocci, diphtheroid bacilli, and *Bacillus proteus*—but *Bacillus vaginæ* is not at all constantly present, though it may be found in some cases. This type of vaginal secretion continues to be present until puberty, when it again changes. The change actually precedes the onset of menstruation; indeed, menstruation can be predicted through this observation, as was proved by Soeken and confirmed by the Japanese worker Miura. Once again the discharge becomes abundant, creamy white in colour, cheesy or pultaceous in consistence, strongly acid in reaction, and again grows *Bacillus vaginæ* in pure culture. In the normal healthy virgin this type of discharge continues throughout the whole period of the reproductive phase of life. At the menopause the discharge again changes, reverting to the type found during childhood—scanty, thin, greatly lowered acidity and a multiple and varied flora of organisms. The explanation of these changes is most interesting and throws great light on the production of vaginal discharges. The work of Cruickshank and Sharman is monumental, and I shall quote from it freely. These workers showed that important histological changes occur in the vaginal mucosa at these periods of life. Not only structural changes, but also biochemical alterations are found, and these changes have been shown to be controlled or produced by the presence in the blood of the female sex hormone, variously named ovarian follicular hormone, theelin or œstrin.

The mucosa of the vagina of the new-born infant and also of the fœtus after the seventh month consists of a thick, florid, stratified, squamous epithelium of many layers, in the cells of which glycogen is abundantly present. After the first week of life notable changes are found, the epithelium becoming progressively thinner and less florid and the glycogen considerably diminished in amount and distribution, so that in infants a month old the epithelium is thin and atrophic and glycogen is uniformly absent.

At puberty the epithelium again becomes thick, florid and rich with glycogen and remains so until the menopause, after which the mucosa becomes familiarly atrophic, its epithelium thin and keratinized, and glycogen either very scanty or completely absent. The cyclical alterations in the mucosa strongly support the contention that they are produced through the agency of the female sex hormone, œstrin, which also is present in the blood in varying concentrations, showing a similar cyclical rhythm.

During the latter months of pregnancy œstrin is present in considerable concentration in the maternal blood, being produced by the Graafian follicle, the *corpus luteum* and the placenta. It is also present in considerable quantity in the amniotic fluid, colostrum and urine. Thus the fœtus is richly supplied with this hormone. After parturition the maternal œstrin rapidly diminishes, while the infant is suddenly separated from its source of supply and after the fifth day of life œstrin is absent from the infant's urine. Œstrin remains absent throughout childhood, but again at puberty we have the maturation of the Graafian follicle, ovulation and menstruation, all depending on cyclical variations in the œstrin content of the blood, which in its turn is dependent on the activity of the anterior pituitary gland. These conditions continue for thirty years or so during the reproductive phase of life, but at the menopause œstrin again disappears, and this marks the end of all sexual cyclical changes.

Many interesting clinical conditions can be fully explained by the variations of the œstrin content and also by the associated resulting changes in the vaginal mucosa.

Thus in the new-born infant engorgement of the breasts, so often seen both in males and females, but twice as common in the latter as in the former, is due to their stimulation by the high œstrin content in both the maternal and fœtal blood at the time of birth.

Another condition often seen is the blood-stained vaginal discharge occurring a few days following birth. This is explained by the sudden withdrawal of the œstrin excess and is analogous to menstruation, which in the adult is explained by the sudden and very considerable lowering of the threshold for the female sex hormone.

A very interesting clinical condition is the occasional case of intermenstrual pain or *Mittelschmertz*. This is sometimes accompanied by very slight bleeding and is analogous to œstrus in the lower animals (Hartmann). The pain is considered to be due to factors concerned with the process of maturation of the Graafian follicle and subsequently ovulation. In other cases at this time there is an excessive vaginal discharge of normal characteristics. Thus in a patient of mine:

Miss Q.K., aged thirty years, complains of intermenstrual left-sided lower abdominal pain for the last three or four years. The onset is always fourteen days following the first day of the last menstruation period. For two days preceding she has a slight white discharge which continues for another three days. On examination she is *virgo intacta* and rectal examination shows that the pelvic organs are normal.

It is well known that in the adult the normal vaginal secretion is not only highly acid, but also highly bactericidal. *Bacillus vaginæ* grows in a highly acid medium containing lactic acid, the result of probably both bacterial and enzyme fermentation of the vaginal glycogen. It is interesting to note that lactic acid is present in the vagina of the new-born infant before the *Bacillus vaginæ* is found.

Cruickshank and Sharman have investigated the reactions of various discharges and express them in terms of hydrogen ion concentration represented logarithmically by the pH value, neutral being 7·0 and figures below that indicating acidity. Later, in dealing with various discharges, reference will be made to such values and also discharges will be referred to under the classification adopted by the Germans, who describe three types of bacterial flora or three grades of cleanliness. Thus:

Grade I: *Bacillus vaginæ* pure culture, creamy white, cheesy discharge, desquamated vaginal epithelium. pH 4·0-4·4.

Grade III: Döderlein's pathological flora. Staphylococci, enterococci, colon bacilli and diphtheroids, profuse or scanty discharge, watery or purulent, less acid and even alkaline. pH 5·6-7·6.

Grade II: *Bacillus vaginæ* and other types above mentioned. Intermediate. pH 4·6-5·6.

The normal vaginal discharge at the periods of life previously described may now be further classified thus:

Birth and first week.—Grade I: Strongly acid and highly bactericidal.

Children.—Grade II: Scanty discharge, faintly alkaline, with varied flora.

Adults of healthy virgin type.—Grade I: High lactic acid content and strongly bactericidal.

Menopause.—Both Grade II and Grade III discharges.

One very interesting fact noted is that during pregnancy discharges which in the early months are of Grade III or Grade II types tend to change in the later months, and in many cases actually become Grade I at the end of pregnancy. For the same reason the normal secretion greatly increases at this time. In other words, under the influence of the increased œstrin content, glycogen is further deposited, lactic acid produced, and Döderlein's bacillus gains the ascendancy. Hence the great risk of disturbing this proteetive mechanism through using douches, and antiseptics during pregnancy.

Anatomical Considerations.

Before proceeding with the practical side of this paper I shall just briefly refer to some other anatomical considerations. Thus Skene's glands and Bartholin's glands do not reach full histological development until puberty and thus are not liable to become infected by the gonococcus during childhood. On the other hand, however, we have seen that at this period of life the vagina, with its atrophic mucosa lacking in glycogen, its walls in apposition, and with an alkaline secretion of Grade II type, is an excellent culture tube for pathological organisms, and hence the frequency of gonococcal vulvo-vaginitis of children with the ease of infection and the resulting epidemics. But great hope is now being placed in a new treatment of this pathetic scourge. As a result of the knowledge of the action of œstrin in developing the vaginal mucosa, this hormone is now being given both orally and subcutaneously to children without any harmful effects and with most encouraging results.

Again, at puberty with full development of the uterus we have the establishment of the cervical secretion, a clear mucus like the white of an egg, alkaline in reaction, pH being about 8·5, and, as will be seen later on, appearing to increase in quantity from various causes not yet fully understood and in the absence of any infection. And, lastly, at the menopause, when all the genital organs atrophy, we have the same liability to infections of the vagina as was present during childhood, such a condition as senile adhesive vaginitis being frequently seen in practice.

Leucorrhœa is a very common symptom, most frequent in married women and complained of in about one-third of the gynæcological patients admitted to a public hospital. In most cases, after thorough examination the ætiology is obvious, but in a large and very important group it is not, and here a complete examination so often fails to dis-

close any real cause. Unfortunately, however, a cause is often assumed, treatment is undertaken, and with results that are, to say the least, disappointing. As an example may be mentioned the leucorrhœa of non-infective origin so commonly seen in adult virgins.

Classification of Leucorrhœa.

Broadly speaking, leucorrhœa may be classified as infective or non-infective in type.

Infective conditions are those due to gonorrhœa, trichomonas, monilia, syphilis, tuberculosis, chancroid, streptococcus, *Bacillus diphtheriæ*, colon bacillus, staphylococcus, *Bacillus welchii, granuloma inguinale*.

Non-infective conditions causing discharge are: carcinoma of Fallopian tubes, uterus, vagina and vulva; sarcoma of uterus, for example, the *sarcoma cervicalis botryoides*, fibroid tumours of the uterus, uterine displacements, including prolapse, also attrition ulcer; pyometra, the result of a partial gynatresia; polypi, both corporeal and cervical; lacerations of the cervix, erosion of the cervix, including the congenital erosion of Fischel; relaxed vaginal outlet; *adenomatosis vaginæ*, a rare condition described by Bonney and Glendinning; urethral caruncles; foreign bodies, both in cervix and in vagina, for example, ring and bar pessaries and the various contraceptive appliances that have come into fashion; diabetic conditions; leucoplakia and kraurosis of the vulva; thread worms and round worms; obstetric conditions, such as vesicular mole.

In addition, among the regional pathological conditions causing discharge I should like to mention particularly the following conditions: intermittent hydrosalpinx — *hydrops tubæ intermittens* and *profluens*; pyometra—partial gynatresia causing intermittent purulent discharge; primary chancre of syphilis on the cervix; various caustics used in the vagina by herbalists; diabetic vulvitis; all contraceptive appliances and applications, particularly the gold spring pessary; stimulation of Bartholin's gland, the result of sexual excitement; conditions of anus and rectum, the discharge from which must be differentiated from the vaginal discharge.

Regarding intermittent hydrosalpinx, this is an interesting condition, the possibility of which has long been debated, but I have at present a definite case under my care.

Following on an attack of acute salpingitis and pelvic peritonitis, which was treated conservatively, this patient is now comparatively well. Her only disability is periodic pain in the right lower part of the abdomen, which is followed by a gush of clear watery fluid, whereupon the pain subsides. During the period of pain the enlarged tube can be easily palpated, but in the intervals little abnormality can be felt. A lipiodol salpingogram would confirm the diagnosis.

A catarrhal condition of Bartholin's duct frequently occurs during the honeymoon and is often followed by a copious discharge of mucous when the tension behind the obstruction is overcome by the accumulated secretion.

You will note that in the above list I have purposely omitted two conditions, namely, chronic endometritis and subinvolution. Reference to these will be made later.

Clinical Examination.

I now come to the consideration of various features of the clinical examination ,after which I shall fully discuss the following common conditions: (i) *Trichomonas vaginalis* vulvo-vaginitis, (ii) monilia infections (mycoses), (iii) gonorrhœal discharges, (iv) non-infective leucorrhœa of virgins.

The clinical investigation of leucorrhœa entails not only a searching inquiry into the history and habits of the patient, but also it is essential to realize that in order to diagnose the condition accurately there must be a spirit of mutual confidence and understanding, gained only through a sympathetic demeanour and tactful interrogation. It seems superfluous to add that a full history and examination are just as necessary in these cases as would be considered necessary in, for example, a case of hæmaturia.

Another avenue to be explored is the delicate, but very necessary, interrogation concerning the husband's health.

Before the gynæcological examination it is better that the patient refrain from any form of douching for forty-eight hours, and she should be instructed not to empty the bladder later than three hours before the examination. This is absolutely essential in cases of suspected chronic gonorrhœal urethritis. When an examination has to be made in the absence of these precautions, and as a result the diagnosis is doubtful, repeat the examination after instructing the patient as to these precautions before giving any final diagnosis. The position of the patient is of importance. While avoiding unnecessary exposure, particularly in single women, a proper exposure and a comfortable position should both be obtained. This, I am of the opinion, is best carried out in the dorsal position, the patient's feet resting on stirrups, the legs encased in leggings, and a sheet placed across the knees, so that the examiner is out of sight to the patient. In single women, inspection of the external genitalia and a rectal examination can be easily carried out so, but if a detailed vaginal examination is required anæsthesia is necessary and is much more satisfactory. Often, however, inspection of the external genitalia will be sufficient to suspect the diagnosis. In all cases the first essential is to see the discharge and next to determine where it is coming from. Therefore all orifices must be inspected and gently massaged to obtain a specimen of secretion. Having obtained the discharge, test its reaction to litmus paper, make films for staining, and examine a fresh drop as described later. Films are stained by Jensen's modification of Gram's method.

With the patient in the dorsal position, the type of vaginal speculum that I have found most convenient is one after Graves, made in three sizes. If in doubt about a discharge which appears to come from the

uterus, place a tampon against the cervix for twenty-four hours and examine it after withdrawal. Other useful points when diagnosis is in doubt are: (i) complement fixation test to reveal metastatic gonorrhœa, for example, an arthritis in the presence of a chronic cervicitis; (ii) urea test when urine is suspected; (iii) injection of sterile milk into the bladder to locate a urinary fistula; (iv) a catheter specimen of urine should be obtained after examination.

I have on several occasions in cases of urethritis found a colon bacillus as the infecting organism and a coexistent vaginal discharge showed the same organism. In obtaining vaginal discharge from children I use a fine glass pipette with a rubber suction cap.

Trichomonas Vaginalis Infections.

Among the infective types of leucorrhœa probably the most common met with is that caused by the flagellate protozoon known as the *Trichomonas vaginalis*. Although first described as present in the vagina and as a cause of vaginitis by Donne in 1837, it received scant recognition until recent years, and now gynæcologists have come to regard the condition as the most common infective type of leucorrhœa. This protozoon is a normal inhabitant of the human body, being present in bowel and mouth and other passages. In what circumstances it causes a pathological condition is not fully understood, for other organisms are always found to be present in a typical trichomonas vaginitis, particularly streptococci and colon bacilli. In any case, however, we do know that the symptoms persist until no trace of trichomonads is found in the vagina. The organism is oval or pyriform in shape, but is also often present in the form of a rounded cyst. It varies in size from that of an epithelial cell to a pus cell, has four flagellæ, an undulant membrane and axostyl. It is very actively motile, but motility soon ceases in the cold and in the presence of even weak solutions of numerous antiseptics.

The disease is rare in children, but is found in adults of all ages, both single and married. It frequently follows a gonococcal cervicitis and occasionally both organisms are present at the same time. This modifies an observation I had made in a previous communication.

The symptoms and signs of *Trichomonas vaginalis* vulvo-vaginitis are almost always very typical. The outstanding feature is discharge — thin, acrid, yellowish green or yellow, frothy and extremely offensive. The pungent odour is characteristic. It is highly irritating and many patients have a discoloured elliptical area of moist, sodden, reddened skin extending from vulva to the anus, due to irritation and intertrigo. The condition is worse following menstruation and even after treatment tends to relapse at this time. The discharge is sometimes sanious. Its reaction to litmus is strongly acid and its pH 5·0 to 5·6. Other symptoms are vulval irritation and pain, dyspareunia, dysuria, and the picture of a general neurasthenia soon develops.

Examination reveals in addition small dark red tender areas of the mucosa of the vestibule and *labia minora*, inflamed-looking *carunculæ myrtiformes* and a peculiar dark red mottling of the vagina and cervix, the latter being referred to by Kleegman as the strawberry cervix, and this may be mistaken for an erosion or a cervicitis.

Diagnosis can be made only by recognizing the parasite, which is best done by examining under the microscope a fresh drop on a warmed slide diluted with an equal volume of warm normal saline solution. It can also be seen in specially prepared and stained films.

I recently saw a patient who complained of a discharge first noticed eight years ago. She had a bilateral salpingectomy performed, but the discharge persisted. A year later total hysterectomy was performed, but the discharge still persisted. She douched herself on and off for a few years, when I saw her and found a trichomonas infection. After a few treatments she was cured.

I could give many instances of failure to suspect this condition, but perhaps Greenhill's caustic comment is still adequate, namely:

That there are still many gynæcologists who have not seen a case of *Trichomonas vaginalis* vaginitis, the chief reason being that they have not properly looked for the associated organism.

But the treatment is more difficult than the diagnosis, on account of the tendency to relapse. Three essentials are: (i) careful cleansing of vulva and of all folds and rugæ of the vagina, (ii) application of a suitable antiseptic, (iii) continuation of treatment until no trichomonads are present.

In addition, patients are instructed to make their toilet by directing the hand from the vagina towards the anus. Many antiseptics have been recommended. The technique which I have found is most effective has the following advantages. It is economical for the patient, is of short duration and requires the use of a colourless antiseptic which is non-irritating. The technique is as follows: Three treatments on successive days, followed by three treatments on alternate days, and thereafter on alternate days until the condition clears up and no trichomonads can be found. In any case three treatments are given after the next menstrual period. The average number of treatments required is twelve.

Tincture of green soap is applied to vulva and vagina with the precautions above mentioned and taking care that the posterior fornix receives special attention. After careful drying, hexyl resorcinol in an aqueous solution known as "S.T.37" is applied in the same way. On other days the patient gives herself a douche of lactic acid (B.P.), one drachm to the pint. At this juncture I cannot too strongly condemn the type of douche apparatus which has a vulcanite nozzle. Many cases of vaginitis are produced by unclean and unsterilized nozzles of this type, and many cases of discharge are caused solely through this. I insist on an old-fashioned douche can with a rubber tube and a glass nozzle.

Monilia Infections.

Another interesting condition met with in general practice is that known as aphthous vulvitis and vaginitis, also described as mycotic vaginitis, vaginal moniliasis or simply vaginal thrush. It is, of course, due to infection by a fungus of the monilia group, and over the surface of vulva and vagina are spread the raised white patches with a surrounding zone of hyperæmia so familiarly known as thrush. This condition does not appear to be at all common in this city, but is most frequently seen in women suffering from diabetes and also during the latter months of pregnancy, both these conditions being regarded as important predisposing causes. The work of Castellani, Plass and Hesseltine has thrown great light on this subject. Castellani has cultivated on Sabouraud's medium many types of monilia and yeast fungi, and has been able to classify them. In women he has found the commonest type to be the *Monilia pinoyi*. Clinically these monilia can be recognized under the microscope in the forms of mycelial threads and conidia, either in a fresh drop preparation or on a stained slide when they are strongly Gram-positive.

Plass and Hesseltine have shown that in cases of diabetic vulvo-vaginitis the fungus is almost always present. Although it has long been recognized that relief of the glycosuria rapidly clears up the symptoms in some cases, yet in others it does not do so, and in order to bring about both relief and cure specific treatment to the fungus is necessary. It is granted, however, that the fungus does grow best under the condition of glycosuria. These monilia also grow best in strongly acid media, a fact of clinical importance also, for the discharge from such an infection is also worse just before menstruation and during the latter months of pregnancy, both periods in which the excess of vaginal glycogen brings about a highly acid secretion of lactic acid. Vaginal monoliasis may also occur in children, in virginal adults and even in senile women. The monilia may also be present in the vagina at the same time as the trichomonads. It has now been proved that some cases of thrush in infants may originate as a natal or post-natal infection from the mother's vaginal infection.

The treatment of mycotic vaginitis is now specific—bi-weekly applications to vulva and vagina of a 1% solution of gentian violet, either in aqueous solution or in glycerine. In addition, the patient takes daily douches of a 1% solution of sodium bicarbonate.

Gonorrhœal Infections.

Reference has already been made to gonococcal vulvo-vaginitis in children. Although gonococcal urethritis is sometimes seen in children, it is extremely doubtful if a true cervicitis ever occurs, owing to the rudimentary condition of the cervical glands. In the adult, however, the cervix and urethra are the two sites most frequently involved, and next in order of frequency occur Bartholin's ducts and glands and, lastly, Skene's ducts and glands. The gonococcus has a predilection for columnar cells, whether on mucous membrane or in glands.

There are no special difficulties in the diagnosis of acute gonococcal infections, but it is of the greatest importance to examine all the potential foci in the female. It is of equal importance to remember that the inflamed orifice of Bartholin's duct, described by Sanger as the *macula gonorrhœica*, is not necessarily a sign of gonococcal infection, and also that only about 60% of cases of Bartholinitis are gonococcal in origin.

Chronic gonococcal infections of the cervix are difficult to detect, even though there is a profuse, tenacious, muco-purulent discharge, alkaline in reaction. Many smears must be examined before definite exclusion of the gonococcus can be safely made, and often even then other aids to diagnosis must be invoked. Such are: (i) Smears taken after sensitization of the patient either by (*a*) intradermal injection of aolan; (*b*) subcutaneous injection of gonococcal vaccine, 10 millions; (*c*) topical application with 10% silver nitrate solution. (ii) Cultures. (iii) Complement fixation tests.

Gonococcal vaginitis is extremely rare in women and, when seen, it occurs during pregnancy as a granular vaginitis. The stratified squamous epithelium and the acid medium of the vagina are both inimical to the delicate gonococcus, and it is probable that only when a Grade III flora is present during pregnancy can a gonococcal vaginitis develop. Hence it is most important to realize the futility of obtaining and examining vaginal smears for evidence of gonorrhœa. The discharge in gonorrhœa in females is not always obvious to the patient; sometimes, indeed, there is actually very little discharge, and even in recent infections I have seen cervices perfectly normal in appearance but which yielded smears teeming with gonococci. The usual course of acute gonorrhœa is for the condition to pass through subacute and chronic stages before cure is obtained, but quite a number undergo cure by resolution in three weeks or so.

The principles underlying treatment of female gonorrhœa are regularity in attendance, thoroughness in treatment, and very great patience on the part of both patient and doctor. I should like to mention that my average time taken in obtaining cure is approximately three months. It is important, too, to guard against the extension of the organism to the tubes and uterus, often the result of unwise treatment. The patient must be forbidden to use a douche and every patient with acute cervicitis is instructed to go to bed during menstruation, at which time I give a mixture containing belladonna and *"Liquor sedans"*.

It is important to have a high standard of tests for cure, and treatment should be suspended for three weeks before tests are taken. Those I rely on are: (i) disappearance of all clinical signs and symptoms; (ii) return of the normal type of vaginal discharge; (iii) "negative" smears taken on four occasions, the first three weeks after treatment has been suspended, the second immediately preceding

menstruation, the third immediately after menstruation, and the fourth twenty-four hours after sensitization.

Non-Infective Virginal Leucorrhœa.

Lastly, I come to that very complicated and difficult problem, namely, non-infective virginal leucorrhœa and those other cases which, although not in virgins, can be included in this category. We have all met with such patients—one can easily picture the types—some thin and flat-chested, with unhealthy skin and their long slender chest suggestive of visceroptosis. In others the general make-up suggests some endocrine dysfunction, for they are excessively stout, mentally dull, and the majority complain of such menstrual irregularities as dysmenorrhœa, oligomenorrhœa, and secondary or relative amenorrhœa. In yet another group there cannot be found or seen anything obvious or even a suggestion of abnormality, and in this latter group the leucorrhœa is often intermittent, disappearing and reappearing spontaneously. Various predisposing causes for the condition have been advanced, and it is possible some may exert their influence, directly or indirectly, on the actual mechanism of production of the discharge, which is not understood fully. Such states that have been mentioned are: general neurosis, psycho-sexual neurosis, sexual perversion, fatigue, chlorosis, other anæmias, debility, constipation, malnutrition and even occupational disturbances. The very number of the above and their extremes of influence must impress the observer that the real truth must lie in other directions. And so it is amongst such cases that little can be found on examination beyond the excessive secretion. Occasionally curette scrapings (curettage is definitely contraindicated) have revealed a glandular hyperplasia and the endometrium so often erroneously described in our student days as that of glandular endometritis. This hyperplasia is known to result from excessive stimulation by œstrin—this and other data lead one to look for this as an important possibility in the true ætiology. Also endometritis is a term that is loosely used and which I have no doubt will eventually largely disappear, except perhaps in obstetrics when describing various septic states of the uterine cavity. Indeed, Curtis showed conclusively that infection of the uterine cavity was a rare cause of leucorrhœa.

In non-infective virginal leucorrhœa the discharge is non-irritating (unless intertrigo results from moisture), thick, white or creamy, and smears constantly show a Grade I flora. Its reaction is strongly acid, the pH being 4·2 to 4·7, and such discharge could not possibly be cervical in origin, for this is viscous, alkaline and with pH 8·0 to 9·0.

Cruickshank and Sharman, in investigating these cases, have found two important factors frequently present: (i) X ray examination of the *sella turcica* revealed a shallow fossa, (ii) urinary œstrin was in excess of the normal amount. Other investigations that they carried out were blood sugar determinations and basal metabolic rate estimations, but these did not reveal any variation from normal. They also found that the average age in the non-infective group was five years earlier than in the infective group, which is significant.

Another feature is that the discharge tends to clear up when least expected and then relapses just as unexpectedly.

Treatment of this condition has been most unsatisfactory. Constitutional treatment directed to those predisposing factors already mentioned has failed to achieve any constant degree of success. Fresh air, supervised diet, administration of iron, thyreoid gland, malt extracts, calcium, yeast preparations and vitamins have all had their vogue and their partial temporary success, and for some time I thought vitamins *A* and *D* in "Radiostoleum" capsules were producing brilliant results until I was disillusioned. However, such treatment cannot do harm, but there are two forms of treatment often practised and which are absolutely contraindicated. These are vaginal douching and dilatation of the cervix followed by curettage.

On the assumption that an endocrine dysfunction related to œstrin excess is a vital factor, a rational method of treatment that is suggested is an attempt to control this by means of the controlling hormone of the anterior pituitary, sold commercially under the name of "Prolan" and "Antuitrin S". I do not yet know, however, of any authentic results of sufficient cures by this treatment to warrant its use in cases of leucorrhœa. Theelin is, of course, useless. Experimentally, excess of œstrin, given therapeutically, has produced atrophy and shrinking of the ovary. Remember that the hormone of any gland is incapable of stimulating its parent gland, and at the best is only substitution therapy.

I have often noticed that a yellow discharge of Grade II type occurs both in female children and in adult virgins after a lengthy period in bed, as, for example, after an operation. Also similarly after exanthemata a discharge is often complained of. I have investigated such discharges and found Grade II types of flora with pus cells and epithelial squames. It is possible that in some cases the discharge is caused by the specific infecting organism. These discharges almost all clear up spontaneously when convalescence is complete, although some last a little longer. Local treatment is not indicated for these, but I think that "Adexolin" has benefited some such cases in children, and I would now suggest giving œstrin for those that persist for any length of time.

Conclusion.

In conclusion, I can only predict that the future will show us the exact influences of the female sex hormones in the production of certain types of discharge; but that for the present each and every discharge must be thoroughly investigated and the diagnosis more often established, for I have endeavoured to demonstrate that no treatment at all is often productive of better results than is ineffective treatment based on incomplete diagnosis or guess work.

SEDATIVES IN ACUTE CEREBRAL TRAUMA.

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(From the Department of Surgery, University of Sydney.)

CAIRNS has stated that in restlessness secondary to cerebral trauma morphine should at all costs be avoided.[1] "It depresses the respiratory centre, sometimes excites the patient, and may mask a relapse into unconsciousness." This doctrine is not generally supported by medical practitioners, and indeed one case of restlessness succeeding a fractured skull was reported in Sydney last year to have been treated, successfully, with six injections of 0·015 gramme (one-quarter of a grain) of morphine with atropine. The writer has treated a large series of patients with cerebral trauma during the past eighteen months; morphine was not used in any of these cases, and in no case was the control of restlessness or delirium unsuccessful by other methods. Some clinicians, however, insist on the remarkable effect of morphine in certain cases, and it seemed wise, therefore, to make some experimental inquiry to discover whether the tolerance of the central nervous system for this drug was affected in any way by a blow on the head.

Experimental Observations.

Since controlled experiments in man were out of the question, cats were selected for this investigation,

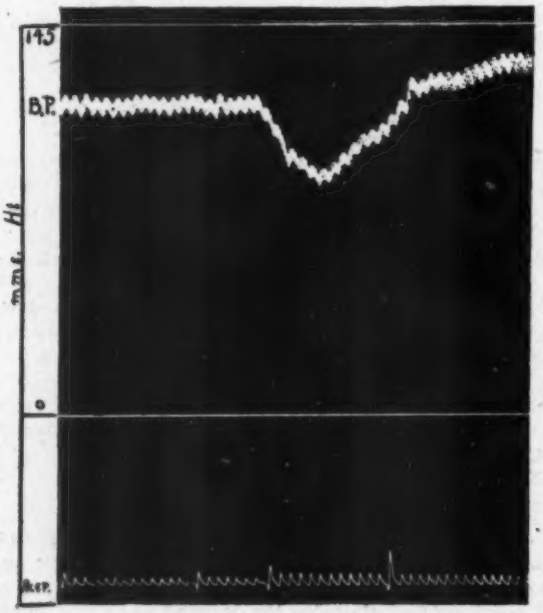

FIGURE I.
Fall of blood pressure and slower and greater amplitude of respiration succeeding traction on the carotid sinus in a "Dial" cat.[1]

[1] In the respiratory records an upward movement indicates expiration and a downward movement inspiration. All records read from left to right. The time of injection of morphine is recorded by the fall of the signal. The time marker records ten second intervals in every case.

and have proved eminently suitable. In the first place the tolerance of the cat's nervous system for morphine is between 50 and 100 times greater than that of man, and one may therefore justifiably assume that any effect produced in the former would be a miniature of that in the latter. In the second place, under conditions of intramuscular injection, a constant dose of morphine per kilogram of body weight of cat produced a constant effect in a series of control animals. It was found that a dose of one cubic centimetre of 4% morphine acetate per kilogram of body weight resulted in comparable modifications of the blood pressure and respiration in a large number of animals. This dose did not suffice to keep the

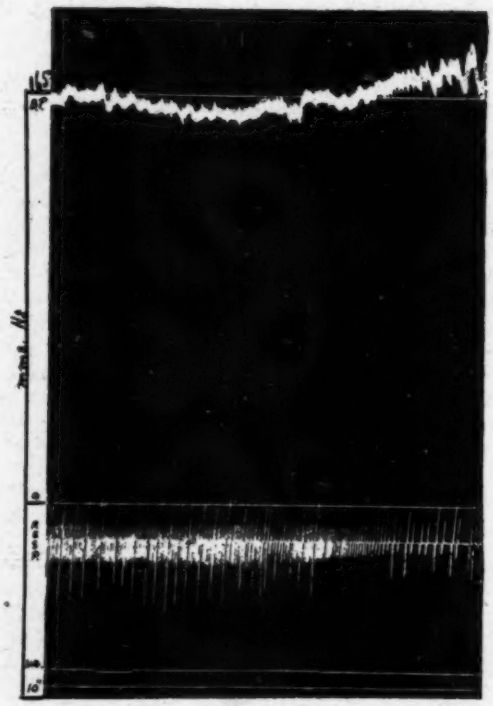

FIGURE II.
Slight steady rise of blood pressure in "Dial" cat succeeding intramuscular injection of 1·0 cubic centimetre of 4% morphine acetate per kilogram of body weight. (In all later figures "morphine injection" implies the same dose as here.)

animal quiet enough for kymographic recording, and premedication by intraperitoneal injection of di-allyl barbituric acid and urethrane ("Dial"-Ciba) in a dose of 0·5 cubic centimetre per kilogram of body weight was required. This latter drug has proved most suitable for the purpose for which it was used, since it acts on the nuclei of the hypothalamus, and in several control animals a normal high blood pressure accompanied by full regular respirations continued for several hours when these animals were submitted to the influence of this drug alone.

A series of sixteen cats has been examined. These have been divided into two groups. In the first

group the combined effects of 0·5 cubic centimetre of "Dial" and 1·0 cubic centimetre of 4% morphine acetate per kilogram of body weight on the blood pressure and respiration of each animal have been studied. In the second group each cat was submitted to the influence of "Dial" and a blow on the head was delivered either by a padded hammer or by a powerful rat trap. Kymographic recording of the blood pressure and respiration was then commenced, and 1·0 cubic centimetre of morphine acetate per kilogram of body weight was injected intramuscularly.

Continuous records were taken for several hours to show any delayed effects of morphine following the head injury and finally the animal was killed and a *post mortem* examination was carried out to determine the nature and degree of the cerebral trauma. Some technical difficulty was encountered when it was attempted to record the blood pressure for long periods from the femoral artery. The lumen of this vessel is very small in most cats, and since clotting proved unavoidable, it was found necessary to use one carotid artery. It was also found necessary when using this vessel to isolate the carotid sinus from the cannula by section of the vessel at a higher level, since if this was not done a marked fall of blood pressure accompanied the tension imposed on the nerve endings in the sinus when any movement of the cat's head took place, or even when the animal made a deep gasping inspiration. (Figure I.)

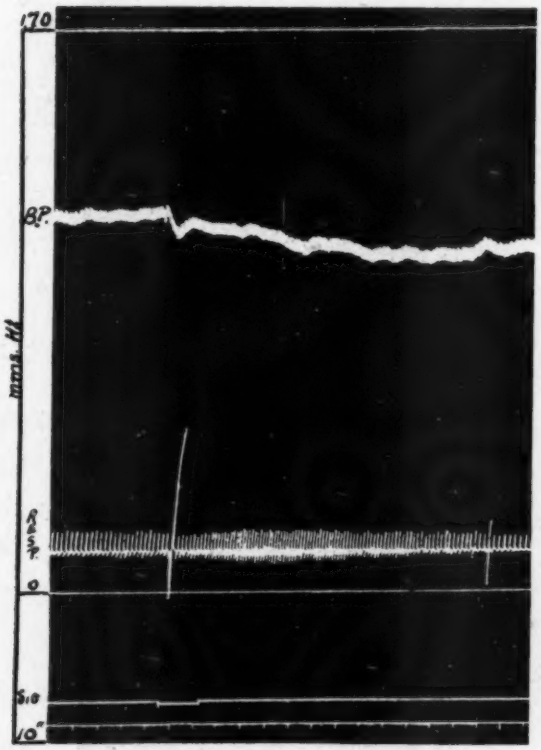

FIGURE III.

Initial increase in the rate and depth of respiration following injection of morphine. The respiratory movements soon become slower and shallower.

The effects of morphine on the blood pressure of a cat treated with "Dial" may be set out as follows:

1. Immediate Effects.

A. Blood Pressure.—There may be a slight initial rise of a few millimetres of mercury lasting for two or three minutes only. No sudden changes even of small amplitude were observed. (See Figure II.)

B. Respiration.—There is usually a slight immediate increase in the rate and depth of respiration. Within two or three minutes the amplitude and rate commences to fall off progressively, and within five minutes these are usually lower than before injection. (See Figure III.)

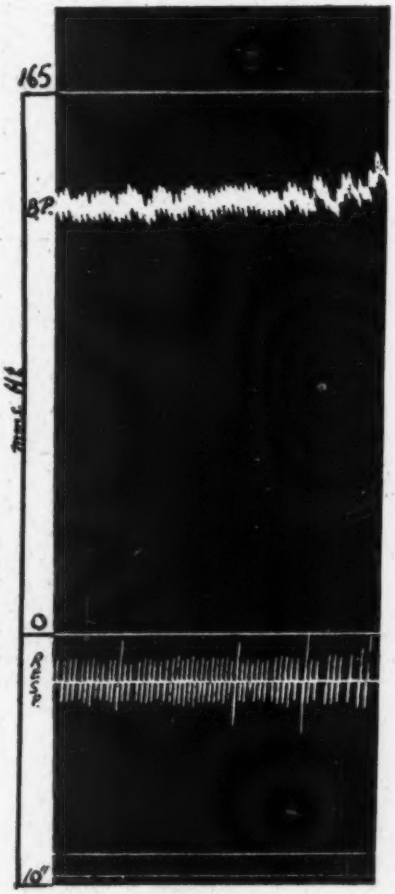

FIGURE IV.

Showing a slight fall (22 mil metres of mercury) of blood pressure fifteen minutes after morphine injection. The original blood pressure record may be seen in Figure II.

2. Delayed Effects.

A. Blood Pressure.—Morphine had surprisingly little effect on blood pressure even when records were kept for as long as five or six hours. The most constant change was a gradual fall of perhaps 20 or 30 millimetres of mercury in the first thirty minutes, and from then on for several hours the pressure kept quite constant in spite of progressive deterioration of respiration. Definite Traube-Hering waves were never produced by morphine alone. (See Figure IV.)

B. Respiration.—Succeeding the initial acceleration progressive slowing of the respiration rate accompanied by increased amplitude and force, chiefly of expiration, took place usually for about one hour. With the dosage used the respiratory centre began to recover after this time, and within one and a half to two hours of injection the rate had increased and the amplitude decreased until the record had regained some of the characters of the original tracing, although a complete return in the original form was not seen. (See Figures V and VI.) Cheyne-Stokes or grouped respirations were never produced by morphine alone.

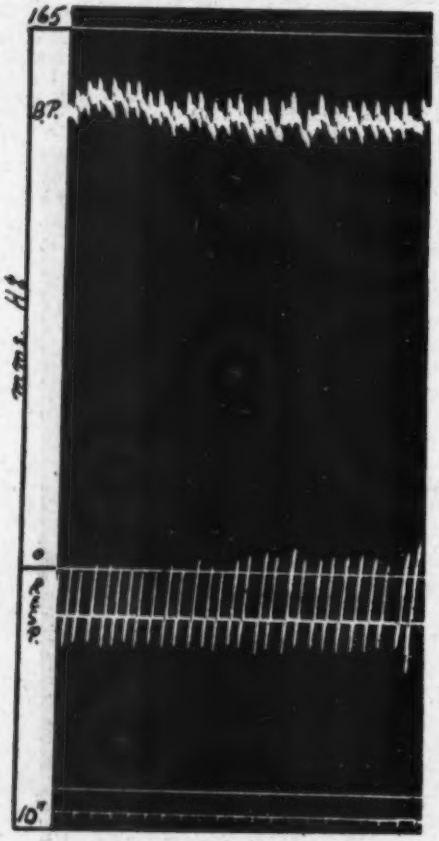

FIGURE V.

High steady blood pressure thirty minutes after morphine injection. The respiratory movements are slow (seven per minute), regular, and the amplitude of expiration is considerably increased. (Compare with Figure IV.)

When the effects of cerebral trauma alone on blood pressure and respiration came to be examined, the constant finding was that a blow on the head was succeeded immediately by a fall of blood pressure of some 20 to 30 millimetres of mercury lasting for about thirty seconds and gradually returning to normal within one or two minutes. (See Figure VII.) At the same time respiration was inhibited for about ten seconds and then recommenced suddenly, of slightly greater amplitude than before but of slower rate. (See Figure VII.) From then on with the relatively mild degrees of trauma used both the blood pressure and respiration might vary slightly from time to time, but the record remained fairly constant for several hours.

When the effects of morphine were combined with those of cerebral trauma the most definite changes were observed, and these may be set out as follows:

1. Immediate Effects.

A. Blood Pressure.—The effect here appeared to depend on the severity of the cerebral trauma. In simple concussion (see Figure VIII) a definite rapid fall of some 20 to 30 millimetres of mercury took place. In more severe trauma (contusion) injection of morphine was often succeeded by a precipitate rise of blood pressure (see Figure IX).

B. Respiration.—The initial acceleration of respiration after morphine was not seen when the drug had been preceded by cerebral trauma. (See Figure IX.) Within two to three minutes of injection (see Figure X) the respiratory movements commence to slow down rapidly, become somewhat irregular, inspiration becomes shallower, expiration more forceful and the movements become gasping.

2. Delayed Effects.

A. Blood Pressure.—Within fifteen to twenty minutes of the injection of morphine, definite Traube-Hering curves develop in the blood pressure record. These curves, which are placed

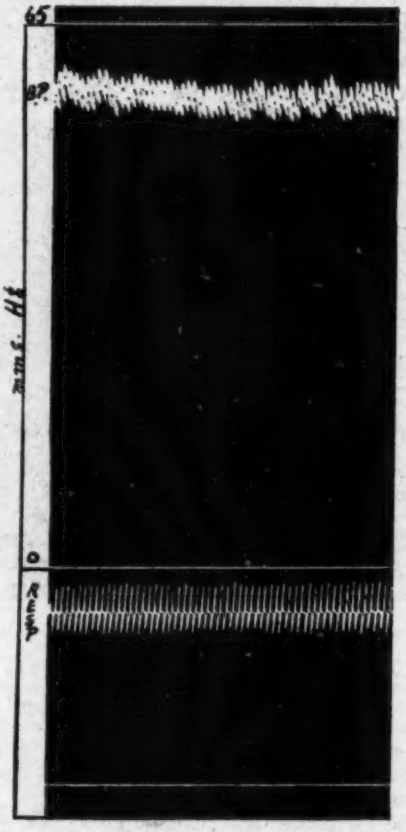

FIGURE VI.

Same record as in Figure V, taken one hour later (one and a half hours after morphine injection). Note the high steady blood pressure and the signs of recovery of the respiratory centre in the decreased amplitude of the gasping expiratory movements and the return to a more normal faster rate.

on a background of fairly high blood pressure, are of considerable amplitude. (See Figure XI.) This type of blood pressure tracing will continue for hours. No animal has succumbed from cardiac failure.

B. Respiration.—In the dosage used the drug was just sublethal to the traumatized respiratory centre. Within fifteen to thirty minutes grouped respirations appear there usually being two respiratory movements in each group. (See Figure XI.) Inspiration is commonly associated only with the first movement of each group. As the record proceeds, the amplitude of the movements becomes gradually smaller and smaller, inspiration being more affected than expiration. (See Figure XII.) In two animals death occurred from respiratory failure, in one case three hours after morphine injection, in the other four and a half hours afterwards.

Discussion.

It can be seen from the above summary that the tolerance of the cat's nervous system to morphine is lowered by a blow on the head. Cushing[2] has shown the relation between a failing respiratory centre, periodic respirations and Traube-Hering waves of blood pressure. Eyster[3] has demonstrated the association of Cheyne-Stokes or grouped respirations and Traube-Hering waves. These prodromal manifestations of respiratory failure were not produced by morphine alone, but appeared under the combined effect of morphine and cerebral trauma. This enhanced depressant action of morphine on the respiratory centre following cerebral trauma is only one argument against its use in the treatment of restlessness succeeding cerebral trauma. Considered *per se*, however, morphine may constitute the final insult to the patient's hind brain and lead directly to his death. The use of morphine is most irrational in these cases on general principles alone.

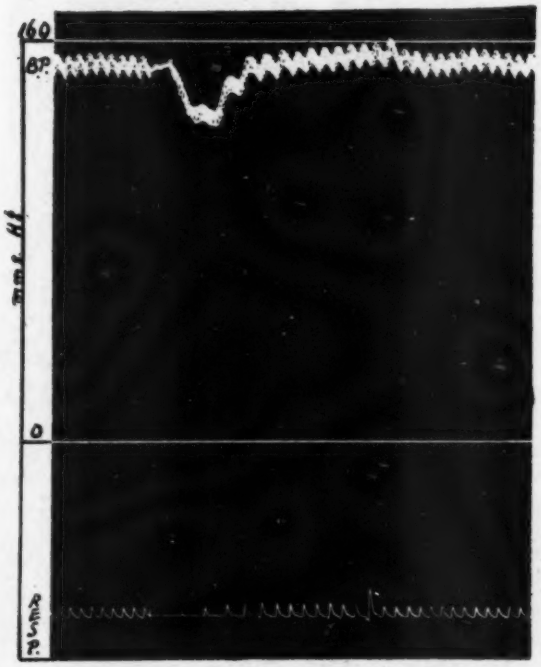

FIGURE VII.

Fall of blood pressure immediately succeeding a blow on the head (cerebral concussion only). At the same time the respiratory movements are completely inhibited, but recommence abruptly within twenty seconds at a larger amplitude and a slower rate. The time intervals in this record are the same as in all the others.

Bard,[4] Fulton and Ingraham[5] have described "sham rage" reactions in laboratory animals in which the temperament of these animals has been transformed by bilateral superficial incisions into the inferior surfaces of the frontal lobes of the brain close up to the mid-line. After this operation there were present in these animals in addition to gross autonomic changes such as the erection of hairs, dilatation of the pupils and sweating of the feet; complete changes in temperament, and rage reactions to stimuli which previously were pleasurable. "Gentle stroking of the animal's back stirred it to a fury of scratching, spitting and attempting to bite. Any restraint

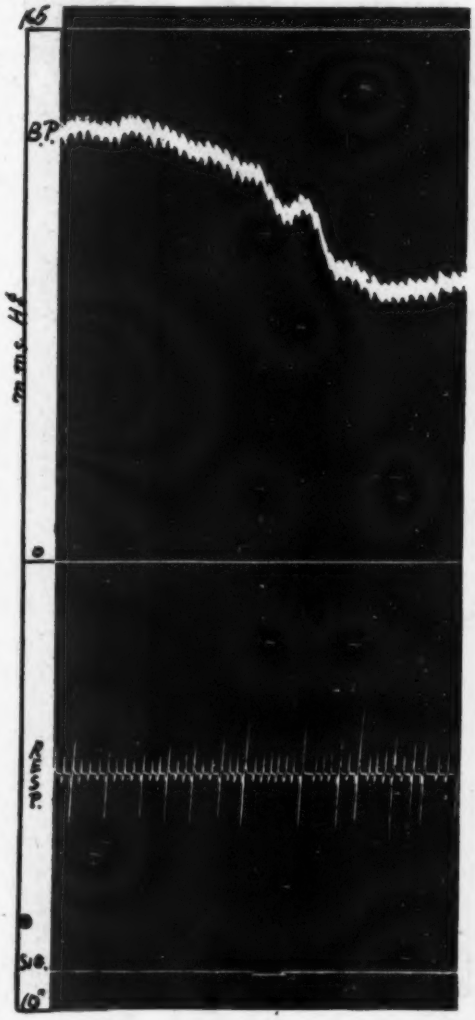

FIGURE VIII.

This animal was concussed by a blow on the head. The heart rate was 160 per minute before the trauma and 112 afterwards. The respirations were 36 per minute before and 26 afterwards. Twenty minutes after concussion morphine was injected, and the record shows the resulting fall of blood pressure. The respirations were at first but little affected.

caused it to scratch and bite, whereas it formerly only purred under the same circumstances." These effects were considered by the authors to be "release phenomena" due to the cutting off of prefrontal inhibitory control of the thalamus and the autonomic centres in the hypothalamus. A patient with a

FIGURE IX.

This animal received a more severe cerebral injury. The resting heart and respiration rates under "Dial" alone were 173 and 28 respectively. After trauma, which proved *post mortem* to be cerebral contusion, the heart and respiration rates were 236 and 14 respectively. The record shows the precipitate rise of blood pressure and the depreciation of respiration succeeding morphine injected ten minutes after the receipt of the blow on the head.

high blood pressure, a fast pulse, profusely sweating, with dilated pupils and either aggressively resisting restraint or curled up in the posture of "cerebral irritation" ready to produce an excessive response to any external stimulus is so much the human counterpart of the laboratory animal with a bilateral inferior frontal lesion that the resemblance cannot be ignored. It is interesting to note, too, that the inferior surfaces of the frontal lobes suffer the greatest damage as the result of a blow on the head (Jefferson[6]), and that restlessness succeeding cerebral trauma is characteristically associated with inferior frontal laceration, contusion or œdema and with

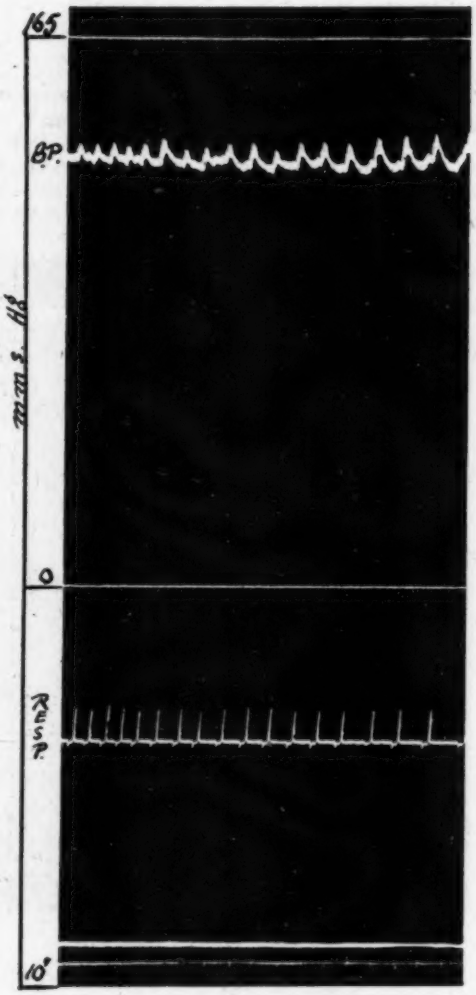

FIGURE X.

The record shows the effect of morphine injection on the respiratory centre of a cat suffering from cerebral concussion. This record was taken five minutes after morphine injection, and the slow rate, shallow inspirations and the deep gasping expirations are to be noted.

sympathetic "release phenomena". Some degree of cerebral œdema is always present in this condition, so that the rational attack on traumatic cerebral restlessness is cerebral dehydration by intravenous injections of either 50% solution of glucose or 15% solution of sodium chloride. If this measure is not wholly successful, then some sedative drug may be required, but certainly not a drug such as morphine, which has a selective action on vital centres in the

hind brain. The drug that is obviously required is one which has a selective action on the diencephalon which has been released from prefrontal inhibition. Such a drug exists in "Dial"-Ciba (di-allyl barbituric acid and urethrane). Its efficiency as a sedative in traumatic cerebral restlessness is at present unknown, but this matter is now under investigation.

FIGURE XI.

Traube-Hering waves develop on a background of high systemic blood pressure eighteen minutes after injection of morphine in a cat suffering from cerebral contusion. Note the irregular grouped respiratory movements, each group occurring at the summit of a Traube-Hering wave.

Summary.

1. The selective depressant action of morphine on the respiratory centre is enhanced by preexisting cerebral trauma.

2. Morphine in a dose having but little effect in a normal animal will produce signs of respiratory failure if it is preceded by even a slight degree of cerebral trauma.

3. The early treatment of traumatic cerebral restlessness should be causal by cerebral dehydration and not symptomatic by drugs.

4. If sedatives are required in addition, a drug should be selected which acts primarily on the diencephalon and not on the vital centres in the hind brain.

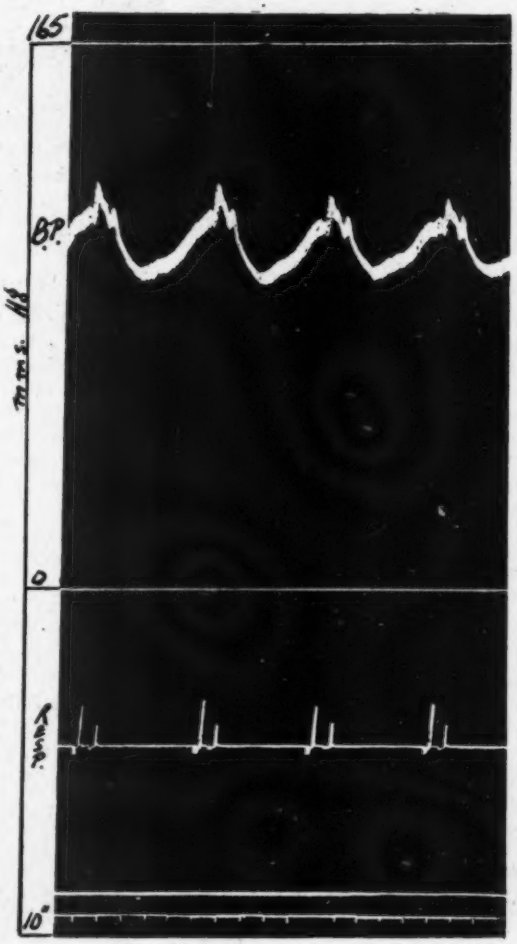

FIGURE XII.

The same record as in Figure XI, taken 105 minutes later (two hours after morphine injection). Note the persistent Traube-Hering waves and the high blood pressure. The respiratory movements have further depreciated and now inspiration is an extremely weak and shallow movement. The amplitude of the expiratory gasps has also diminished. The death of this animal occurred one hour later. No gross intracranial hæmorrhage was revealed *post mortem*.

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THE PRACTICAL METHOD OF DEALING WITH ASTHMA AND HAY FEVER.[1]

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ASTHMA.

THE days are past when the physician shook his head before the asthmatic patient and said that nothing could be done except giving an antispasmodic mixture, to be taken regularly, and giving adrenaline during attacks. Every patient with asthma should be investigated along certain prescribed lines in order to determine the cause, if possible, and, the cause or causes being determined, to outline appropriate treatment. It is my object to omit theory as far as possible and to outline the practical approach to treatment through investigation.

Types of Asthma.

The contraction of the bronchiolar musculature, together with the associated œdema of the mucous membrane which constitutes asthma, is produced both by specific and non-specific causes. It is well, then to have some definite classification of asthma on which to work. No classification is perfect, but I find the following of most use from an ætiological viewpoint.

1. Nervous.
2. Hyperpietic.
3. Asthma with upper respiratory infection.
4. Asthma with mechanical nasal defect.
5. Allergic: (*a*) inhalant, (*b*) bacterial, (*c*) food, (*d*) drug.

Frequently an asthmatic individual can be classified under two or more of these types.

Type 1.—The purely nervous type of asthma, which is a spasmodic hyperpnœa, is rare. This is included here for completeness of classification and, further, because of the nervous factor (so-called) in the majority of asthmatic individuals.

Type 2.—The hyperpietic group of asthmatics is a small one. These people are probably all allergic in type, but have a high blood pressure reading. They are placed in a group by themselves because blood pressure readings are low in the majority of asthmatics.

Type 3.—Type 3 refers to the asthma which is caused reflexly by antral and sinus infection. It is often associated with a bronchitis.

Type 4.—Type 4 includes those who have polypi, septal spurs, and enlarged turbinates, all of which may reflexly cause attacks of asthma.

Type 5.—Type 5 includes the large group of asthmatics who are specifically sensitive to foreign substances, whether inhaled or ingested, or who harbour pathogenic organisms causing a bronchitis or a local tissue damage.

Investigation.

Clinical History.

The clinical history of the patient is of great importance. This must be taken in detail, with reference to the time of attacks—whether they occur at certain seasons of the year or in certain localities. Seasonal asthma suggests a pollen sensitivity. When the attacks occur in certain localities, some substance in the environment is suggested, and details must be obtained of such possibilities—horse dander, flour, kapok, feathers *et cetera*. Often the patient volunteers the information that dust, turning beds, feather pillows, aspirin *et cetera* will produce attacks. This must not prevent the physician from carrying out the complete investigation, however, as there are often other causes which are unsuspected by the patient. Digestive disturbances may be suggested by the history. The duration of attacks is often helpful, since attacks lasting up to one week or more, particularly occurring in the winter months, are often due to sepsis or an underlying bronchitic infection. In these patients there is usually a history of purulent sputum during attacks.

Physical Examination.

A complete routine physical examination must be carried out.

Special Examinations.

The following special examinations should be made: (i) A careful examination of the ear, nose and throat by a specialist is advisable, but in any case a thorough investigation of the nose and accessory sinuses is essential, preferably with an X ray examination of the sinuses. (ii) Chest X ray examination for enlarged hilar lymph nodes or any other abnormality. (iii) Test meal in many cases is recommended. (iv) Skin testing for evidence of hypersensitivity.

Skin Testing.

Skin testing is not the simple procedure that it might appear to be, and the results must be considered carefully in conjunction with the clinical history of the patient. Reactions vary in intensity at different sites on the skin surface, and it is advisable to select the same site in each patient. The forearm, being the most convenient, is selected for performing the tests.

Food sensitivity is not uncommon in the first decade, but after that inhalants are of most importance. Multisensitivity is not infrequent, and here again careful attention to the history of the patient is of great importance in determining the cause of the attacks.

A few patients show an erythematous reaction to the slight trauma occasioned by the test, due to a liberation of histamine-like bodies in the skin; if left for about half an hour and if care is exercised in applying the test substances the tests can be completed. It is not advisable to attempt any skin tests during an attack of asthma or following an injection of adrenaline, as these may lead to false negative reactions on the skin. Positive skin reactions occur in about 80% of asthmatics. Freshly prepared extract must be used, and all skin tests must be carefully controlled. It is advisable not to attempt more than a dozen at a time.

1. *Cutaneous Scratch Method.*—In using the cutaneous scratch method, cleanse the forearm with

[1] A post-graduate lecture delivered in Sydney, May, 1935.

spirits. Shallow incisions, three millimetres long, are made with a clean dry scalpel in the skin, insufficiently deep to draw blood. These are placed at least 2·5 centimetres (one inch) apart and are numbered as desired with a skin pencil. One cut is used as a control, to which is applied Coca's solution only, and the test substances are applied to each of the other cuts. When dry reagents are applied, a drop of Coca's solution is first put on the cut and the powder is applied next with a loop or applicator and gently rubbed into the cut. When extracts are used for testing they are applied directly to the cut. The results are read within twenty minutes, and a positive reaction shows an area of erythema of varying extent at the site of application of the allergen, with or without wheal formation.

2. *Intradermal Method.*—In using the intradermal method, about 0·01 cubic centimetre of the extract to be tested is injected into the skin to produce a minute wheal. A similar quantity of Coca's solution is injected into another area to serve as the control. Results are read in twenty minutes. A positive reaction is an area of erythema, usually with the presence of a wheal.

3. *Patch Method.*—The patch method consists in the application of the test substance to the unbroken skin for a period of twenty-four hours. This method is employed usually for skin testing for foods. A paste of the fresh food is made up with Coca's solution or $^{n}/_{100}$ sodium hydroxide solution and spread on a piece of lint about 2·5 centimetres (one inch) square. This is applied directly to the skin and covered with oiled silk, which is strapped to the arm. A positive reaction consists of a wheal or local erythema at the site of the patch after twenty-four hours, when the control of the diluting fluid shows no response.

Skin Testing Reagents.—Skin testing reagents in common use are: (i) pollens—Kentucky blue grass, plantain, capeweed, dahlia, sunflower, Iceland poppy, cosmos, wattle, paspalum, sweet pea; (ii) animal emanations—horse dander, cat hair, dog hair, duck feathers, cattle hair, sheep wool, rabbit fur; (iii) dusts *et cetera*—house dust, kapok, orris root, linseed, le Page's glue; (iv) foods—wheat, egg white, milk, fish, tomato, honey, chocolate, peanut, green pea, lettuce; (v) bacterial antigens.

These constitute the main substances for skin testing, and extracts of the epithelia, dusts and pollens are prepared in capillary tubes for skin testing by the Commonwealth Serum Laboratories. When testing for foods it is best to use the fresh reagent wherever possible. Many further tests may be performed when the history suggests them.

When recording the results it is convenient to use a standard such as "+" for an area of erythema up to one centimetre in diameter, "++" for an area from one to two centimetres, "+++" for any larger area, and "++++" for a wheal over five millimetres in diameter surrounded by an erythematous area.

When the skin tests are read the arm should be cleansed with spirits.

The Cause of Attacks.

Contact with a specific allergen will precipitate an attack, for example, proximity to horses in a horse dander-sensitive patient. There are, however, many non-specific causes which produce attacks in the asthmatic individual, such as steam, perfume, westerly winds, laughing, sneezing, physical exertion, overloaded stomach *et cetera*.

Diagnosis.

There is little difficulty in the diagnosis of asthma. There is frequently a history of other allergic manifestations in the individual, such as hay fever, urticaria or eczema, past or present. In the majority there is a family history of allergy in the present or previous generations. So-called bronchitis in infants and children with a family history of allergy is usually asthma if it occurs frequently.

Positive skin reactions place the patient in the allergic or potentially allergic group of individuals. Asthmatics of the bacterial type often give no response to the scratch tests when the inhalants or ingestants are used, but a small injection of vaccine may produce a marked attack of asthma lasting many days.

Prognosis.

The prognosis depends upon whether a specific sensitizing substance or substances can be found, and the elimination of these substances from the environment. Failing that, the desensitization method of injections will give temporary or permanent relief from attacks. It follows from this that seasonal asthma, which means a pollen sensitivity, is most likely to show the best results.

Those patients showing marked lung fibrosis and bronchitis, whether sensitive to inhalants or not, offer the least promise of complete relief, unless a suitable climate and vaccine can be found for them.

Treatment.

General Treatment.

The confidence of the patients must be obtained throughout the investigation and treatment, also their intelligent cooperation as well in excluding possible irritants.

Excess physical exertion producing undue fatigue must be avoided, together with emotional disturbances, as these are liable to produce attacks.

Overloading the stomach at any time is harmful, particularly a heavy night meal, and for this reason it is best to have the large meal of the day at midday. Indigestible foods, such as fatty foods, bananas, cucumbers, pork and condiments, should be omitted from the diet.

The bowels should be kept well opened with salts if necessary.

A breathing exercise of the slow, free expiratory type should be carried out for five minutes twice daily. Standing with the arms extended from the side of the body, in line with the shoulders, bring the arms slowly to the front till the hands meet, at the same time let the air flow out slowly and easily through the open mouth without any noise. Maintain this position of expiration for a couple of

seconds. Then swing the arms out to their original position smartly, breathing in through the nose rapidly, with the mouth closed. There should be just a comfortable emptying and filling of the chest during this exercise. This exercise is most valuable for all asthmatics. Dental caries should be properly attended to and infected tonsils removed, but I have not yet seen the removal of tonsils cure an asthmatic individual. Enlarged adenoids causing obstruction should be removed. Antral infection must be treated conservatively, as major operations on the accessory sinuses often make the patients considerably worse. Polypi in the nasal cavity should be removed.

The Acute Attack.

Adrenaline is the best treatment in the acute attack and should be given in 0·3 to 0·6 cubic centimetre (five to ten minim) doses of 1 in 1,000 solution. If given in small doses, it should be repeated. If the attack is severe, morphine and atropine sulphate should be given.

Strong black coffee, caffeine citrate or a dose of antispasmodic mixture may be sufficient to abort a mild attack.

Status Asthmaticus.

Small injections of adrenaline, 0·12 to 0·18 cubic centimetre (two to three minims), repeated every couple of minutes, should be given through a needle left in position in the arm. Morphine and atropine in small doses are necessary. The removal of half to one pint of blood from the arm may be advisable in certain patients. Chloroform anæsthesia or "Amytal" narcosis (0·6 gramme or ten grains in divided doses) is of value in some patients. It is advisable to give 0·3 gramme (five grains) of calomel by mouth, no matter how bad the patient may appear, as soon as possible.

Specific Treatment.

The elimination or avoidance as far as possible of the substances to which the patient is sensitive is most desirable. Feather pillows, mattresses and eiderdowns should be avoided by those showing skin positivity to feathers. Foods which cause attacks should be eliminated from the diet. In many cases, when these harmful substances are avoided, the asthma will stop without further treatment.

Densensitization by Extracts.—To those sensitive to dust or grass pollens and other irritants in the environment which cannot be removed, a course of desensitizing injections with an extract of the substances must be given. These extracts are obtained from the Commonwealth Serum Laboratories and are given by subcutaneous injection, beginning with the weakest strength, "A", and proceeding on to "B" strength up to "D" strength. A guide to this scheme of injections is as follows:

"A" strength:	0·1 cubic centimetre.
	0·2 cubic centimetre.
	0·4 cubic centimetre.
	0·8 cubic centimetre.
"B" strength:	0·1 cubic centimetre.
	0·2 cubic centimetre.
	0·4 cubic centimetre.
	0·8 cubic centimetre.
"C" strength:	0·1 cubic centimetre.
	0·2 cubic centimetre.
	0·4 cubic centimetre.
	0·6 cubic centimetre.
	0·8 cubic centimetre.
"D" strength:	0·1 cubic centimetre.
	0·2 cubic centimetre.
	0·3 cubic centimetre *et cetera.*

The injections are given twice weekly till the optimal dose is reached, that is, the dose which will keep the patient free from attacks. Large local reactions on the arm (over 7·5 centimetres or three inches in diameter) or a general reaction within a few hours, that is, an attack of asthma following the injection, calls for a more gradual increase or even a reduction of the dose. It is obvious that an overdose has been given when an attack occurs after a few minutes of injection of the extract. On account of the large reactions, both local and general, it may not be possible to reach the "D" strength. When the optimal dose is reached the injections are given at less frequent intervals, say, once each week for a few weeks, then once each month for three months. In the seasonal or pollen-sensitive individual the injections must be commenced well before the season begins, in order to reach the optimal dose before pollination begins. It is advisable to give three pre-seasonal courses of injections with pollen extract. Alternatively, an injection may be given every month or second month throughout the year. In dealing with the perennial type of inhalant asthma, such as that due to house dust, a periodic maintenance injection of house dust extract is all that is necessary. It must be understood that the perennial type of asthma may also be due to an underlying bronchitis or infection which may complicate a dust sensitivity.

Autogenous Vaccines.—In the bacterial type of asthma, and when infection complicates the inhalant type, autogenous vaccines should be used. It is best to begin with a very weak dilution (say 50 millions per cubic centimetre) and a small quantity (say 0·1 cubic centimetre). One injection per week is quite sufficient; the quantity is increased by 0·1 cubic centimetre per week. A stronger dilution is used when 1·0 cubic centimetre of the weak vaccine has been given. Overdosage will precipitate severe attacks in the bacterin-sensitive individual, and the attacks begin one to three days after the injection. Where a bacterin sensitivity complicates an inhalant sensitivity the vaccine may be given concurrently with the inhalant extract.

Non-Specific Treatment.

Non-specific treatment should be used only where no specific sensitivity has been found following exhaustive tests, unless the history and examination point to radiation or tuberculin treatment.

In regard to non-specific treatment the following should be noted:

1. Peptone injections of Armour's 5% solution are given intravenously.

2. Tuberculin injections act well in tuberculous patients with asthma and must be given in very minute doses at first (say 0·1 cubic centimetre of a

dilution one in ten millions). The dosage is increased very gradually. In non-tuberculous patients it acts well in some instances.

3. Stock vaccines are of value and should be used with the same precautions as the autogenous vaccines.

4. Deep X ray radiation is of most value in the children and young adults who have enlarged hilar glands.

5. Change to a dry climate from the coastal districts in bronchitic patients is sometimes the best treatment, especially for the very young children.

6. Antispasmodic mixtures should be given until desensitization treatment can bring some measure of relief.

7. "Luminal" in doses of 0·06 gramme (one grain) once to three times daily is valuable.

8. Dilute hydrochloric acid should be given to those who have an achlorhydria, and may be given as a therapeutic test to patients who complain of indigestion and flatulence. It is given in 2·0 to 4·0 cubic centimetre (half to one drachm) doses with water at each meal time.

9. Glucose in 15·0 cubic centimetre (half ounce) doses night and morning is valuable in many patients, especially children.

10. Ephedrine causes unpleasant symptoms in many patients.

HAY FEVER.

Hay fever frequently precedes asthma (about six in ten patients). Careful inquiry concerning the history of the hay fever often helps in the investigation of the asthma.

Classification of Hay Fever.

Hay fever may be classified as follows:

1. Seasonal, usually due to pollens.

2. Perennial: (*a*) due to inhalants which are constantly present in the environment of the individual, such as dust, feathers, horse dander *et cetera*; (*b*) due to chronic infections which may be of a low grade toxicity. In this subgroup skin tests produce no reactions.

These groups may be combined in the same individual, when pollen or other inhalant sensitivity may be associated with a chronic low grade infection.

Investigation.

A careful clinical history is as important here as before. Examination of the nose and accessory sinuses for gross infection or structural change is essential.

Skin tests are performed in exactly the same manner and with the same substances as in the investigation of asthma. Tests may be carried out also by applying a small quantity of the substance to the mucous membrane of the nose. The interpretation of the results of the skin testing must be as careful also.

Treatment.

1. Desensitization to inhalants is carried out by injection of extracts as in the scheme previously outlined. Pre-seasonal injections are given for three consecutive years in the case of pollens. Overdosage causes irritability of the nose and eyes. The patient should avoid flowers to which a sensitivity is shown, for example, decorating the house with dahlias or Iceland poppies when these give a positive skin reaction.

2. Vaccine therapy, either by autogenous or stock vaccines, is of value in the infective type. More rapid increase may be made in the dose of injections than in asthmatic patients, but an overdose may precipitate a severe attack of hay fever.

3. Conservative treatment to the accessory sinuses is recommended.

4. An ephedrine nasal spray is of value until the desensitizing injections have had effect. Saline nasal douches should not be used on account of the irritation of the mucous membrane which is produced by frequent usage.

5. For relief of an acute attack, painting the mucous membrane with adrenaline, ephedrine or cocaine is the best form of treatment.

Reports of Cases.

SCHILDER'S DISEASE.

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WITH A PATHOLOGICAL REPORT BY
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ENCEPHALITIS PERIAXIALIS DIFFUSA, diffuse sclerosis or Schilder's disease, is a condition of sufficient rarity to justify the presentation of a fairly classical case.

The disease was originally described by Schilder in 1912,[1] and a sufficient number of cases have subsequently been recorded to establish it as a clinical entity.

In a review of the literature published in the *American Journal of Diseases of Children*, June, 1930,[2] 78 cases were collected from the world's literature. The first case fully described in Australia appears to be that published in *The Lancet* of October 26, 1929, by de Crespigny and Woollard, of Adelaide.[3]

In a recent monograph (1934) by Professor Bouman, of Utrecht,[4] approximately 100 cases are collected.

The history of the patient upon whom these observations are based is as follows:

A male child, aged seven years, was admitted to hospital on April 5, 1934. He had been a bright boy at school and was perfectly well until November, 1933, when he began to complain of headache, and it was noticed that he used to run into obstacles and had become clumsy in his movements. He became emotional and displayed violent fits of temper, his speech became slow, and his eyesight began to fail. At this time also he developed visual hallucinations, seeing "fires and savage wild animals".

Examination of his eyes showed his sight approximately $^6/_{60}$, with fields of vision markedly limited. His pupils reacted normally to light, and no changes were observable in the optic disks, although fine pigmentation was recorded in the retina. Nystagmus was present with slow component to the right side.

Hearing was defective, although he could apparently appreciate loud sounds.

He sat up continually in bed, gibbering in an insane manner about "lions, tigers, fires and machine guns", saliva dribbling continuously from his mouth.

Neurological investigation revealed marked spasticity of lower limbs, with general increase of tendon reflexes. Plantar response was extensor on both sides.

Cerebro-spinal fluid was normal, except that the globulin content was increased. The Wassermann test gave no response.

FIGURE I.
Patient, male, aged seven years. Note attitude and position of hands, arms *et cetera*, and general apathy.

A clinical diagnosis of Schilder's disease was made; the patient gradually passed into a condition of complete dementia and died on May 28, 1934, approximately six months from the onset of symptoms.

Macroscopically the *post mortem* findings were irrelevant, and the brain was given to Dr. Oliver Latham, Pathologist to the Mental Hospitals Department, New South Wales, for investigation. The pathological findings amply confirmed the clinical diagnosis and will be briefly reviewed.

In the accepted pathological conception of the disease, as recently described in an admirable monograph by Bouman, of Utrecht, the following features are described:

1. Large patches in the brain, particularly in white matter; outstanding feature of these patches is disappearance of myelin sheaths. This process extends throughout vast territories. As a rule one single patch is distributed through both hemispheres, the two halves being connected by a demyelinated part of the *corpus callosum*.

2. Smaller patches are commonly found distinct from these large patches. A striking feature is that the patches often stop at a short distance from the cerebral cortex; a thin layer of white matter between the cortex and the patch is usually spared, although the degenerative changes may encroach on the cortex. In most cases all these patches are clearly visible in the fresh section of the brain, but in others they appear only in specimens in which the myelin sheaths have been stained for microscopical examination.

3. The myelin sheaths remaining are of the thin type; all the thick sheaths have disappeared. Those still present show many varicose swellings, particularly in those sheaths situated just outside the borderland of the patch.

4. In other parts of the central nervous system disseminated sclerotic patches are present: in the brain stem, cerebellum and spinal cord. The glial tissue always shows progressive alteration with regard to both macroglial and microglial cells.

5. The proliferated macroglial cells constitute many glial fibrils, but the felt-work proceeding from this reaction is as a rule less dense than that seen in disseminated sclerosis.

6. The proliferated microglial cells become spherical (scavenger cells, "*Gitterzellen*") and are seen laden with breakdown products.

7. In nearly all cases the blood vessels inside the patches are proliferated and around each vessel the reticular connective tissue has grown out into a wide meshed network, lying in the nervous parenchyma.

8. Further, in the sheath of most medium-sized blood vessels a well marked infiltration, with many lymphocytes, some plasma cells, numerous scavenger cells, fat granules, macrophages and mast cells, is found.

Diagnosis during life may be arrived at when the cardinal symptoms are manifest. For example, a healthy child developing central blindness, deafness, with mental deterioration leading to complete dementia, and exhibiting a varying degree of spastic paraplegia, presents a clinical picture peculiar to this disease.

The condition has to be differentiated from: (*a*) cerebral tumour (particularly in unilateral cases), (*b*) encephalitis, (*c*) acute disseminated sclerosis, (*d*) cerebral syphilis, (*e*) juvenile paralysis (general paralysis of the insane), (*f*) amaurotic family idiocy (Tay-Sach's disease).

PATHOLOGICAL REPORT.

(O. LATHAM.)

The brain and part of the cord of a child seven years of age, reputed to have died of Schilder's disease, was handed into the laboratory, preserved in 10% formalin solution. On section no abnormality could be made out with the naked eye, save that the pia-arachnoid was somewhat thickened and opaque.

It has been our good fortune to have examined many forms of myelitis, including eight cases of disseminated sclerosis (two being of the acute variety), four cases of encephalomyelitis, many cases of *tabes dorsalis*, of the combined scleroses, of the myelitis following various forms of injury, germ infection, hæmorrhage, of the myelitis associated with various developmental anomalies, and especially of that form called "compression myelitis" or primary degeneration of the neurone. In the last mentioned one finds those peculiar myelin sheath dilatations and axis cylinder fusiform swellings (torpedoes) which, with many of the pathological entities found in multiple sclerosis, also enter into the pathology of Schilder's disease.

A piece was therefore excised from each side, including some of the isle of Reil, external and internal capsule. The piece was mordanted, sectioned in paraffin, stained according to Weigert's technique, and counterstained with azo-fuchsin G. and picric acid. At once there was evident a peculiar bilateral sclerosis and demyelination, something between encephalomyelitis and multiple sclerosis. Also certain "zones" could be made out. By this we mean that almost any stain revealed a sort of core of almost pure microglia in *Gitterzellen* stage and sometimes apparently nothing else, that is a microscopically fluid centre. Special stains, however, revealed many blood vessels, probably including new formations, and a fine interstitial material. This interstitial material became more evident as one proceeded outwards, where the microglia became mixed up with an ever-increasing number of macroglia cells (gemastete cells) and with innumerable glia fibres almost as dense as in multiple sclerosis. It was now seen (and was subsequently proved by Ziehan-Cajal's gold sublimate stain) that the fine interstitial tissue was really glial fibres from these macroglia cells. No axis cylinders or myelinated fibres could be made out in this dead centre.

As we proceeded outwards the microglia sensibly diminished, while up to a certain point the macroglia became more dense and important, presenting different pictures according to the position occupied and the con-

ILLUSTRATIONS TO THE ARTICLE BY DR. A. HOLMES À COURT AND DR. OLIVER LATHAM.

FIGURE II.
Cut surface of formalin-hardened brain, apparently normal macroscopically. Dotted ink line on both sides shows the "proved" areas involved in the sclerosis; the cortex outside these lines is normal. Doubtless the sclerosis involves other areas not included in this picture. (× 3/4.)

FIGURE III.
Section taken through and internal to island of Reil (C). Arcuate fibres (A) beneath cortex, spared and staining darkly, showing sudden transition from normal to demyelinated area (W.S.). Anterior end of caudate nucleus reveals the anterior limit of the demyelinating process. A few swollen myelinated fibres in transverse section have taken the stain darkly. All the pale area here should be jet black. Weigert's myelin stain. (× 4.)

FIGURE IV.
Section shows a "V" gulf which is the lower end of the descending horn of the lateral ventricle. The darkly staining pyramidal area is a gyrus of occipital lobe, internally; the rectangular side represents the white matter bounding the ventricle. Normally this area should stain jet black—myelinated fibres. Actually scarcely 2% are visible. Weigert's myelin stain. (×.)

ILLUSTRATIONS TO THE ARTICLE BY DR. A. HOLMES À COURT AND DR. OLIVER LATHAM.

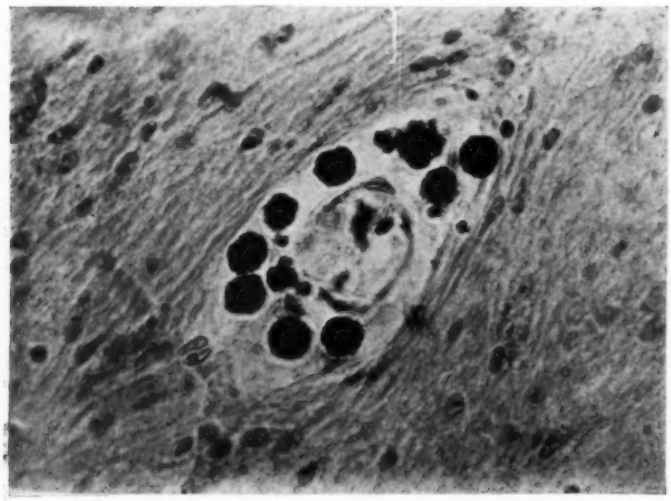

FIGURE XI.

FIGURE XI and FIGURE XII.

Very large *Gitterzellen* (Collier and Greenfield's large globoid cells) in perivascular spaces, the purple granules of which make them stain darkly. Nissl's method, with cresyl violet. Large macroglia also prominent around vessels in Figure XII. (× 400.)

FIGURE XII.

ditions present (pressure ?). The result was that sometimes these macroglial cells in great numbers lay compressed with most of their fibres running parallel. Myelinated fibres now increased in numbers until normal conditions obtained, macroglia ceased to be conspicuous, and there appeared to be no involvement of the grey matter, as would occur in a tumour of such extensive dimensions. Indeed, one of the most conspicuous features of the myelin-stained preparation was the sharp demarcation of the lesion at the arcuate fibres, which, being left intact, made a conspicuous black line arching round the base of the gyri to separate the main lesion from the normal cortex.

Outside the lesion's centre scattered myelinated fibres appeared, and these were often swollen and stained poorly or stained and gave up the stain too readily. The swelling was irregular, revealing alternate lengths of the fibres, thick to thin fusiform swellings. The axis cylinders also showed similar alterations in calibre, being also twisted, scarcely staining at all (yellow by Cajal's silver method) or faintly dark or properly black. When efficiently stained the diseased centre could readily be distinguished from the normal by the naked eye.

Pieces were now taken from the rolandic and other parts of the cortex, and especially from round the lateral and third ventricles on both sides, from the pons, medulla and spinal cord. They were prepared by use of the following stains and methods: (i) hæmatoxylin and eosin, iron hæmatoxylin and picro-indigo-carmine; (ii) our modified Nissl's stain and also cresyl violet and neutral red; (iii) frozen sections were stained by Scharlach R for the fats *et cetera*; (iv) Cajal's silver stain for axis cylinders as well as Bielchowski's stain; (v) the Ziehan-Cajal's gold sublimate method for macroglia; (vi) various myelin stains with and without picric acid-azo-fuchsin.

With these sections at our disposal we were able to make out almost all the lesions laid down by Professor Bouman as common in Schilder's diffuse sclerosis. We were able to determine that the focus of greatest demyelination and destruction lay around the hinder end of both lateral ventricles (the so-called "storm centre"). This may have been the starting point of the condition.

The process spread forwards and upwards to include territories beneath the isle of Reil and motor cortex bilaterally, but stopped or became far less intense as one reached the caudate nucleus, which had escaped damage.

It has been said that one case differs from another in clinical signs, sight being most affected in one patient, smell in another, limb movements *et cetera* in a third. It is not necessary to postulate a great difference in the area of reaction, for I am assured that function could be quite good in many parts of the diseased brain tissue. What probably does matter is the position and extent of the fluid centre. Here function must surely be in absolute abeyance, except where certain tracts of myelinated fibres can survive, apparently intact, in the middle of quite affected parts.

It remains to be added that we found other typical softenings in the brain stem and dorsal part of the pons and a remarkable and typical anatomical degeneration or loss of probably 80% to 85% of the ordinary myelinated fibres of both pyramids in the pons and spinal cord. The last mentioned lesion is remarkable in that the loss of myelinated fibres must have been of old standing, in that we failed to perceive one *Gitterzell* to suggest an active or at least "present" process, and also no very obvious gliosis of special type. This is extremely rare in disseminated sclerosis, and this was true of both crossed and direct pyramidal tracts and of the vestibulo-spinal tract. These lesions amply supply reasons for the observed clinical signs, and we must add that actual softening occurred as far forwards as the descending pocket of the third ventricle, near the optic chiasma. The rich assortment of perivascular cells in the lesions were well brought out by Scharlach R, with which some intracellular granules stained a definite scarlet and some only yellow—further instances of the variation of staining observed in this disease.

Perhaps the best picture of these huge perivascular *Gitterzellen* (the large globoid cells of Collier and Greenfield) was obtained by using cresyl violet, and the axis cylinder peculiarities were shown with Bielchowski's stain. No doubt many neurones (nerve cells) were destroyed in the main lesions; we noted many diseased. No specially diseased neurones, however, could be made out apart from the lesions or in the cortex. The pia was thickened, a little infiltrated; and subpial felting was evident, as well as gliosis of the first cortical layer.

Bouman observes that no direct connexion can be made out between interstitial reaction and parenchymatous loss.

Nothing distinctive could be made out in the cerebellum or neurones in the cord. By shutting off light or utilizing over-silvered preparations the macroglia can be shown to be associated with an extraordinarily rich and fine glial fibre formation. From the lesions in the cord it seems obvious that the fluid centre certainly includes part of both internal capsules. This little fact alone separates this affection from disseminated sclerosis. The lesion in no respect resembles giant-celled astrocytoma of the cerebral hemispheres. Very little actual dead tissue is evident—there has been no "sudden destruction or death"—one is not reminded of gummatous granulomata. No tabetic affection of the cord or cord pia is evident; the complete sparing of the cortex and the absence of perivascular gliosis and of mononuclear infiltrations, apart from the lesions, rules out other luetic affections, and the apparent preservation of the cortical neurones excludes Tay-Sach's disease.

Neither by the naked eye nor microscopically can the lesions be said to favour a vascular distribution; this is rather against encephalomyelitis.

In what way may these observations negative certain suggestions regarding the ætiology of diffuse sclerosis?

Vascular lesions can be ruled out. Active infection would surely give us more of the picture obtaining in known infections.[3] Anatomical degenerations are often absent and in any case are not extensive enough for the primary lesions, thus resembling multiple sclerosis. Bearing in mind the remarkable differentiation and richness in processes of the microglia and oligodendroglia as revealed by modern staining processes and their extraordinarily intimate connexion with the myelin sheath, one is constrained to listen to those who postulate an hereditary and/or acquired deficiency of function towards the myelin metabolism, perhaps in connexion with these interstitial tissues of the nervous system, the oligodendroglia in regard to myelin nutrition, and the reticulo-endothelial microglia in their phagocytic activities and ability to transform disintegrated products into transportable substances.

A most interesting discussion dealing with many aspects of demyelination, linking up tabes, combined sclerosis, Tay-Sach's disease, encephalomyelitis, disseminated sclerosis, various toxic processes associated with scattered myelitic foci *et cetera*, will be found in Davison's article, wherein he deals with the clinical signs and pathology of a disease very similar to "diffuse sclerosis" occurring in several baboons and monkeys. With due diffidence he inclines to the idea of that error in lipoid metabolism such as appears to occur in xanthomatosis.

SUMMARY.

The clinical history and neurological signs of a seven-year-old boy with many of the accepted signs of Schilder's disease have been presented, together with a report of the histological examination of the brain and cord. These revealed many of the accepted pathological lesions distinctive of this disease.

ACKNOWLEDGEMENTS.

Acknowledgements are due to Dr. I. H. Sender, of Kurri Kurri, New South Wales, who referred this patient for investigation, and to Miss V. Jones, of Sydney Hospital, for excellent production of photomicrographs.

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DIFFUSE "ENCEPHALITIS", PRESUMABLY SCHILDER'S DISEASE.

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IN view of recent interest in Schilder's disease, otherwise known as *encephalitis periaxialis diffusa*, or probably more correctly as diffuse sclerosis, it has been decided to put on record this brief clinical history. We understand that our colleagues at Sydney Hospital were recently fortunate in obtaining *post mortem* material confirming a diagnosis of Schilder's disease. We await with interest the publication of their complete pathological investigation, and can only regret that in this case it was impossible to obtain an autopsy. Whilst realizing this very great drawback in attempting to record what is admittedly a rarity, we feel that a neurological condition which was obviously bilateral, occipital and slowly progressive to a fatal termination warrants this diagnosis.

B.B., a girl, aged ten years, was referred to one of us (J.F.) by Dr. Dwyer, of Gosford, on October 3, 1934, with a history of dimness of vision. The mother stated that the child had had whooping cough for "about three weeks" and that on September 21, 1934, she had noticed that the child had difficulty with her vision. Further questioning in hospital elicited the fact that her Sunday school teacher had thought her vision to be affected "a week or two" earlier than this date, thus, if this is accepted, dissociating the visual defect from the pertussis. On examination on the first occasion (October 3, 1934) she could see hand movements only with each eye. The ocular movements, pupil reactions and fundi were normal; in the absence of general clinical signs these findings made it difficult to postulate an organic basis for the condition. The patient was advised to report immediately in the event of further developments, but in any case not later than one month.

When the child was seen again on November 9, 1934, the mother thought that it could see a little better. On examination the vision in each eye was only perception of light and the fundi were normal. The right eye was a little divergent and the left eye was turned slightly upwards. The pupils were dilated and reacted sluggishly to light. At this interview the mother gave some information that may or may not be significant, by stating that about a month prior to the first visit the child had said she saw double about three times. Arrangements were made for her admission to the Royal Prince Alfred Hospital forthwith.

On examination by one of us (T.M.G.) on November 14, 1934, there were no localizing neurological signs other than the almost complete blindness. She had some perception to strong light. The child was somewhat excitable, and the mother thought that she was a little more so and more loquacious than before the commencement of her illness. She was afebrile and looked well. The pupils, though dilated, reacted to light, though not briskly, and the eye movements appeared normal. A provisional opinion that her case was one of Schilder's disease was recorded, subject to full investigation of the cerebro-spinal fluid.

Lumbar puncture on the following day and again at a later date revealed clear fluid with no increase in pressure. There was no reaction to the Wassermann, Kline or any of the chemical tests. Lange's curve showed no deviation from the normal. Other investigations, hæmatological, urinary and radiographic, were normal.

After five weeks in hospital the child was removed to her home. Slight ptosis (left) was noted on December 21, 1934. She had become somewhat irritable, at times drowsy, and had vomited on one or two occasions. She had been afebrile except for slight pyrexia following lumbar puncture.

For the record of her subsequent progress we are indebted to Dr. Dwyer, who writes as follows:

> Unfortunately this patient died on February 8, 1935. After leaving Royal Prince Alfred Hospital she was seen by me on January 2, when her condition was evidently the same as on her discharge. On January 5 we admitted her to Roma Private Hospital because she developed severe headache, was extremely restless and refused all food. On January 7 she developed generalized convulsions during which (the nurse stated) she assumed an opisthotonic position. These were the only convulsions during the course of the illness. From then on she became more restless and developed incontinence of urine and fæces. We had to keep her on bromides continually. She showed no further focal signs than you were aware of. Twenty-four hours before her death she suddenly became comatose.

Comment.

Other provisional diagnoses were naturally considered, one prominent in the discussion being encephalitis due to a neurotropic virus associated with whooping cough, such as is sometimes seen after measles and other virus diseases. We have had no experience of whooping cough encephalitis, though the little we have seen of that associated with measles would lead us to expect a more acute encephalomyelitis than we had here. In fact, any inflammatory reaction, no matter how mild (and here we incline to Professor Bouman's[1] conception of demyelination as non-inflammatory), is surely doubtful in view of the completely negative cerebro-spinal fluid. Thus we did not consider that the few and variable deviations from the normal ocular movements were sufficient to establish a diagnosis of the better known lethargic type of encephalitis, although later on in hospital the patient became drowsy during the day and restless at night. Perhaps some of the signs which we are not prepared to explain are among the "highly controversial neuropathological phenomena" which the reviewer of Professor Bouman's book refers to in a recent edition of this journal.[2] It should be noticed that the only constant localizing sign suggested a bilateral occipital lesion. Cerebral tumour or abscess, apart from cerebro-spinal fluid indications, would surely later have shown focal signs. It is to be noted that Dr. Dwyer has said that no other localizing sign occurred and that the child developed generalized convulsions with opisthotonos, probably due to the advancement of the lesion anteriorly. She died on February 8, 1935, her illness thus lasting four months. Collier[3] states that the duration of reported cases varies from seven days to three years, the average being nine months.

In an excellent summary of Bouman's book the reviewer mentioned D'Antona's general statement that diffuse sclerosis was "a subacute affection . . . which is progressive, is found in young people . . . scarcely ever accompanied by fever and exhibiting psychical symptoms that suggest bilateral localization". We suggest that this description fits our case. If, as stated, there have been only two cases reported in Australia, we would hardly claim the third without *post mortem* confirmation; but the clinical history is submitted for its interest.

References.

(1) L. Bouman: "Diffuse Sclerosis (*Encephalitis Periaxialis Diffusa*)", 1934.
(2) THE MEDICAL JOURNAL OF AUSTRALIA, February 9, 1935, page 182.
(3) Frederick W. Price: "A Textbook of the Practice of Medicine", 1934.

The Medical Journal of Australia

SATURDAY, JULY 27, 1935.

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INFANT MORTALITY AND ILLEGITIMACY.

INFANT mortality is described by K. Stourman[1] as "the price of adaptation paid by each generation when entering life, just as the settlement of a new country takes its toll from its immigrants". Stourman, who was formerly Chief Statistician of the Health Section of the League of Nations Secretariat, has given an extensive description of the demographic setting of infant mortality. His description is based on a study carried out in seven European and four South American countries under the auspices of their respective national health services. It will at once be apparent to anyone who sets out to study infant mortality, that he is faced with a fascinating, though complex, problem. A proper appreciation of the present attitude of civilized peoples to infants can be gained only by considering the historical background. This has been well drawn for Australian readers by Professor Harvey Sutton in his Anne MacKenzie Oration of 1931 (THE MEDICAL JOURNAL OF AUSTRALIA, November 14, 1931). Professor Sutton shows how in the past economic and religious ideas were responsible for treatment meted out to infants. It will be clear that, once the economic advantages of a healthy child life had become apparent, social development, with its many inventions and technical improvements, made the conservation of infant life easier. But it needed more than inventions and technical improvements—preventive medicine, its ideals and its practice, came into being and reaped a harvest more bounteous than had ever been anticipated. It would be useless to try to determine how far the reduction in mortality among infants achieved in recent years has been due to social and economic development on the one hand and to preventive medicine on the other. Professor Sutton has said that preventive medicine is the province where medicine joins hands with common sense, but obviously the term common sense could not be applied to any activity that ignored modern inventions and technical improvements.

The general conclusion reached by Stourman is that what he terms the gateway to life has been rendered less perilous, that the world has been rendered less hostile to young human life. He states, moreover, that this amelioration is still in progress and is even being accelerated. He devotes considerable space to the illegitimate infant. Under penalty of paying a greatly increased fee for entry, the infant must present itself upon the threshold of life with two parents duly united in wedlock. The mortality among illegitimate infants varies widely from one country to another. Stourman points out that the illegitimate infant will probably always be subject to a somewhat higher mortality than the legitimate infant, owing to the improvidence of the mother, which is frequently the very cause of its conception. Again, and partly for the same reason, illegitimate births are most frequent in the layer of the population which has been unsuccessful in reaching a normal working class standard of life. A considerable proportion of illegitimate infants will therefore be exposed to unsatisfactory social and sanitary conditions. If the excess mortality among illegitimate infants is to be reduced, society must be ready to remedy this state of affairs. Stourman publishes a map showing the frequency of illegitimate births in European countries in 1929. The Nordic

[1] *Quarterly Bulletin of the Health Organization* (League of Nations), December, 1934.

and Germanic countries show, on the whole, a high frequency of illegitimate births; they are fairly rare in Great Britain and Ireland, the Netherlands, Belgium, Switzerland, Italy and the Balkans. The frequency of illegitimate births, according to Stourman, is low in the United States of America, Australia, New Zealand and South Africa. In 1932 the number of ex-nuptial births in the Commonwealth of Australia was 5,239, or 4·71% of the total births. Stourman's general impression is that psychological factors outweigh racial and social influences in determining the incidence of illegitimacy. He shows that the influence of religion is much less in evidence than has generally been believed. It is curious that no definite relation can be established between the frequency of illegitimacy and the excess mortality of illegitimate infants. Some countries having a high proportion of illegitimate children, such as Austria, Sweden and Portugal, have a low excess of illegitimate infant mortality; at the same time this excess is high in Ireland and Switzerland, where the incidence of illegitimate births is low. Stourman suggests that possibly the social opprobrium resting upon the unmarried mother weighs more heavily in the latter countries than in the former, which would account for the low rate of illegitimacy, but which in its turn reacts unfavourably on the fate of the infant. He finds, however, that this explanation does not hold good for Italy and Bulgaria, where the excess mortality of illegitimate infants is relatively low, in spite of the low frequency of illegitimacy. Only one country in the world can so far boast of having altogether abolished the excess mortality of illegitimate infants. In Iceland the illegitimate infant death rate had in 1928-1929 become 2·5% lower than the rate for legitimate infants. Stourman believes, and most people will agree with him, that there are ways of protecting the illegitimate infant without at the same time encouraging illegitimacy.

Many countries are complaining of a falling birth rate. In this regard it is interesting to note the statement of Stourman that the fall in the birth rate, together with the reduction in infant mortality, has resulted in a far greater demographic efficiency than at any previous time in history. "The new generation is produced and an increase of population maintained with a far smaller effort than before: fewer pregnancies and child-births but more babies surviving." If this is true, the fears of certain publicists must be looked upon as groundless, and woman may claim to have a less burdensome social position. This, of course, leaves untouched the question of illegitimacy and the death rate among illegitimate children. Australia is one of the countries in which the death rate among illegitimate children is not high; it has yet to reach the position achieved by Iceland. Satisfactory laws exist in the several States for the legitimation of children born before the marriage of their parents. Legal enactments of this kind, however, do not affect the death rate of such children. Society needs to take care of unmarried mothers; they cannot fight for themselves—they have neither financial means nor political influence. Stourman is perfectly right when he states that the fate of the infants of unmarried mothers is more dependent upon a quality of which too little is said in our days—namely, the heart culture of the people—than upon the general state of its wealth and sanitation.

Current Comment.

THE TREATMENT OF SPASMODIC DYSPHAGIA.

DURING recent years considerable attention has been directed to two interesting conditions affecting the function of the upper part of the digestive tube. One of these is the strange malady to which the name achalasia of the œsophagus has been applied, the other is the so-called Plummer-Vinson syndrome. The latter has been given many names, and has been recognized for a number of years as being a condition of atrophic change occurring in the pharyngeal mucosa associated with glossitis, achlorhydria and hypochromic anæmia. It is known to physicians that the symptom of dysphagia may be quite satisfactorily relieved in these cases provided the atrophic changes in the pharynx have not progressed too far, that is, if the patient comes under treatment early, is treated efficiently with hydrochloric acid and iron, and persists with the necessary maintenance dose. But it is also known that sometimes too much damage appears to have been inflicted on the tissues involved, and though the blood picture presents a normal appearance, the patient still experiences some difficulty in swallowing. This would perhaps not be a serious matter if no further

tissue change occurred, but, as pointed out by Lambert Rogers in an article on the subject, there is a tendency for these patients to develop carcinoma of the hypopharynx.[1] There seems good reason, therefore, for the persistence of dysphagia to encourage further interest to be taken in the local condition.

Rogers, in collaboration with a laryngologist colleague, thought it would be worth while making an attempt to relax the sphincter and to improve the blood supply. Investigation proved that the sympathetic component of the pharyngeal plexus was derived entirely from the superior cervical ganglion. Accordingly removal of the left superior cervical ganglion was performed in a suitable case, followed after eleven days by the removal of the corresponding ganglion on the right side. The immediate result of this operation was the appearance of a bilateral Horner's syndrome, that is, enophthalmos and miosis, and the patient experienced considerable relief as regards the trouble in swallowing. This patient was a woman, aged fifty-nine years, and œsophagoscopic examination had shown no evidence of new growth, but a cracking of the mucosa behind the cricoid cartilage. She also showed the usual changes in the blood and gastric secretions. Of course it is not known as yet whether the improvement noted during the few months that have elapsed since the operation will be maintained, but Rogers states that he has deliberately sought early publication in order to draw attention to the potentialities of sympathectomy in a condition that may be very dangerous. Trial and experience alone will prove whether the procedure will gain and keep a place in surgery; but it is also most important that all practitioners should recognize the possibility of a cancer occurring in the pharynx of patients (chiefly women) who suffer from this particular type of anæmia.

Though the other form of dysphagia above mentioned falls into an entirely different category, it is worth mentioning here, since attempts have been made to relieve it also by surgical procedures performed on the sympathetic nervous system. G. C. Knight, writing from the research laboratories of the Royal College of Surgeons of England, describes the various types of achalasia, and deals with the means of treatment.[1] He distinguishes between true achalasia, a failure to relax, and true cardiospasm, where there is a definite spasmodic contraction of the muscular structures at the lower end of the œsophagus, and in addition mentions hypertrophic stenosis of the cardia. He has gone very carefully into the question of the exact sympathetic innervation of the part involved, and states that the only adequate method of denervation is to excise the left gastric artery with its surrounding fat and nervous tissue. In this way the leash of sympathetic nerves is completely removed, and evidence of the effect of this procedure was obtained when cœliac sympathectomy was performed in an attempt to treat gastric ulcer by this means. In a case quoted it was observed that two years afterwards food passed with unusual rapidity through the cardia. Knight did not consider that the other operations proposed for the same purpose were equally satisfactory, and accordingly when a suitable case was found the suggested method was tried. In this and in a small series of subsequent cases there is a definite history of relief of symptoms. It is not necessary here to traverse the many procedures, minor and major, that have been used in the treatment of achalasia; the most popular is some form of gentle mechanical dilatation, as with the large bougie advised by Hurst. But here again is a method which will have to stand its trial; there is a great wave of enthusiasm for sympathetic surgery at the present time, and from all the work that is being done will emerge the truth as to the value of the various operations.

[1] *The British Journal of Surgery*, April, 1935.

PSORIASIS.

THE cause of psoriasis continues to be elusive and the more or less empirical treatment unsatisfactory. One of the numerous theories of ætiology is that the lesions are caused by a toxin, arising from some septic focus; another is that there is a local infection by bacteria or fungi. Treatment based on such theories has been employed from time to time. For example, suspensions of psoriasis scales in alcohol or formaldehyde solution have been administered parenterally as vaccines. D. Panja and P. A. Maplestone have recently carried out a series of investigations into treatment and causation.[1] They found intestinal sepsis in a number of cases; but after employing vaccine therapy and other measures for its elimination, they decided that it was not a factor in causation. They found that injection of alcoholic suspensions of scales caused considerable pain, and injection of formalin suspensions caused local necrosis also. Acting on the assumption that psoriasis was a virus disease and that the causal organism was in the corium as well as the epidermis, they ground down pieces of the whole skin, made suspensions with saline solution, filtered, added phenol to the filtrate, and used the filtrate as a vaccine. Injections were given every third or fourth day—intradermally at first, subcutaneously later as the dose increased. Similar vaccines have been used in other skin diseases; they might be employed more extensively with advantage. Some of their patients were apparently cured, no relapse occurring within ten to sixteen months in eleven of twenty-eight cases. They remark that "the theory of a filtrable virus being the cause of psoriasis appears to be well founded". This report is of considerable interest and should stimulate further research; the notorious tendency of psoriasis to remissions and exacerbations should preclude the unconditional acceptance of Panja's and Maplestone's findings.

[1] *The Indian Medical Gazette*, May, 1935.

Abstracts from Current Medical Literature.

PHYSIOLOGY.

On the Inhibition of Gastric Secretion by Oil of Peppermint.

H. NECHELES AND JACOB MEYER (*American Journal of Physiology*, January, 1935) have investigated the effects of oil of peppermint upon gastric secretion in five dogs with Pavlov and Heidenhain pouches, gastrostomies or enterostomies. Oil of peppermint applied locally to secreting gastric mucosa causes a suppression in free acid flow, which is not associated with a relative increase in buffer activity. In a previous communication these authors have described the inhibitory influence of oil of peppermint upon human gastric secretion, using as subjects peptic ulcer patients in whom the acid secretion of the resting stomach was either suppressed or diminished by small doses of oil of peppermint administered by stomach tube.

Effects of Adrenaline Injection in Moderate Work.

THE injection of adrenaline is followed by numerous and interdependent modifications of function. Of the twenty-seven such effects listed by Peters and Van Slyke, perhaps the one which has caused most argument is the influence of adrenaline on carbohydrate metabolism. The increase of blood sugar and respiratory quotient was at one time thought to mean an increase in carbohydrate utilization, but the lactic acid and ketone bodies increase also, and both of these bodies may artificially increase the respiratory quotient, while the formation of ketone bodies implies a decreased utilization of carbohydrate. Thus the view now held is that adrenaline promotes glycogenolysis and may actually retard the utilization of carbohydrate. This view is challenged by D. B. Dill, H. T. Edwards and R. H. De Meio (*American Journal of Physiology*, February, 1935). They have carried out a series of experiments dealing with the influence of adrenaline injections on the utilization of carbohydrate in moderate exercise, and have found that adrenaline, instead of impairing, facilitates the oxidation of carbohydrate in moderate activity. They observed the total nitrogen excretion, the variations in respiratory quotient in blood sugar and blood lactic acid over a twenty-four hour period during which the subject, a fasting man, expended 5,000 calories of energy. The excretion of acetone bodies was also observed. Seven such experiments were carried out on this man—three controls, three with adrenaline and one with glucose. He proved to be an admirable subject, as he exhibited in a high degree uniformity of state from day to day. The injection of adrenaline in three portions of one cubic centimetre each had no effect on protein metabolism, but increased the carbohydrate utilization to more than half what it was during the corresponding period in the control experiments. Smaller quantities of adrenaline produced smaller results. In other subjects the injection of adrenaline had an effect smaller in magnitude but of the same sign. Further evidence that adrenaline, instead of impairing, facilitates the oxidation of carbohydrate in moderate activity is found in the fact that the excretion of acetone bodies was always less after adrenaline than in corresponding periods of control experiments.

The Variability of Non-Hæmoglobin Iron.

BECAUSE of the difficulties inherent in the direct determination of hæmoglobin there has been a widespread interest in the indirect methods of approach. Of these, the oxygen capacity is the most accurate, but the measurement of total blood iron has been preferred by some workers. The accuracy of hæmoglobin values calculated from determinations of total blood iron depends on the assumption that all the iron of the blood is contained within the red blood cells, combined with hæmoglobin, and behaves as a single variable. Theodore G. Klumpp (*Journal of Clinical Investigation*, May, 1935) has found that non-hæmoglobin iron varies widely and is a significant fraction of total iron. From determinations of oxygen capacity and total blood iron in fifty-seven subjects under various conditions he found that an average of 5·3% of the total iron was uncombined with hæmoglobin. He also found that there was no correlation between non-hæmoglobin iron and oxygen capacity. His results indicate that attempts to arrive at values for hæmoglobin by determinations of blood iron are subject to an average error of 5·3%, with a standard deviation of 3·9% and a possible error as great as 17·8%. From simultaneous determinations of total blood iron, oxygen capacity and serum iron it was evident that the serum contained only a fraction of the non-hæmoglobin iron. In this work oxygen capacity was determined in duplicate by the carbon monoxide method of Van Slyke and Hiller. Total blood iron was measured in duplicate or triplicate by volumetric titration with titanium according to the technique recently described by Klumpp.

Basal Metabolism of the Immediate Pre-Adult Years.

MARIAN E. STARK (*American Journal of Physiology*, April, 1935) reports a series of basal metabolism tests on sixty-two boys of ages seventeen to twenty years from the University of Wisconsin and the University High School. This series supplements a previous study of girls of the same ages and permits some generalizations and comparisons between the two sexes. Statistical comparisons of available prediction standards show that the Harrison-Benedict adult prediction extrapolated in a straight line for these ages is the most successful for both series. The present series of men represents an unusually wide range of heights. But probably because the group is too homogeneous in weight for height relationship, there is no significant gain in prediction accuracy when height and weight both are included in the prediction equation over that attained by using weight alone. The importance of including height in formulæ for predicting basal metabolism is not at all as unquestioned as the need for including weight. Dreyer considers height an "erroneous biometric measurement" and achieves an admirable degree of success with a formula which leaves it entirely out of consideration. On the other hand, in the age range up to twelve, the heat production apparently shows more nearly a direct proportionality with height than it does with weight. Du Bois has stressed the desirability of further study of this point. The present study indicates that within the latter half of the second decade the relationship of metabolism to body size is essentially linear in type, though there is distinctly not a direct simple proportionality between heat production and body weight. An example of abnormal body build illustrates the possibility of grave misconceptions that can arise from the use of a prediction standard that makes the latter assumption. These tests were run in duplicate and the Sanborn-Benedict clinical apparatus was employed, with the usual precautions as to the securing of basal conditions.

Adrenal Insufficiency in the Rat.

IN 1856 the adrenal glands were not considered essential for life by Philipeaux, because a high percentage of his rats survived removal of both glands. Since that time many other workers have recorded a low death rate among rats deprived of their adrenal glands. A possible explanation was furnished by Weisel in 1898, when he showed that many of his subjects had demonstrable accessory tissue. On the other hand a number of recent papers report a high percentage mortality following double adrenalectomy. F. J. Agate, junior, and R. L. Zwemer (*American Journal of Physiology*, February, 1935) have attempted to determine the factors responsible for the widely divergent findings for the adrenalectomized rat. In 239 rats of four different strains, bred in the laboratory, the mortality rate after adrenalectomy was less than 10%. The same strains have been reported by others as having an 80% to 100% mortality rate. Operations upon an additional 67 animals derived from outside sources were less successful, the mortality rate for this group being 50%. Consideration of these

results leads the authors to a discussion of the term cortico-adrenal insufficiency. The normal metabolic demands for the cortical hormone seem to be increased by a variety of factors, such as chance or operative infection, traumatized tissue, prolonged anæsthesia, a general "run-down" condition of the animal, or an inadequate diet. Additional factors might be injected toxins or metabolism stimulants, such as the thyreoid hormone. We can therefore have degrees of adrenal insufficiency depending on the ratio between the hormone available and the demand factors. Under optimum conditions of operation and care no excessive demands occur. This allows sufficient time for the accessory tissue to increase functionally so that the hormone available is in excess of normal demand. The authors consider that the excellent living conditions in the department rat colony play an important part in producing a very much lower mortality rate in laboratory rats than in rats from outside sources.

BIOLOGICAL CHEMISTRY.

Coprosterol Formation.

R. SCHOENHEIMER AND W. M. SPERRY (*Journal of Biological Chemistry*, October, 1934) have conducted experiments on bile fistula dogs fed on a sterol-free diet. The fæcal sterols consisted mainly of coprosterol with small amounts of cholesterol and dihydrocholesterol, as in the case of the normal dog. Thus dietary or bile cholesterol is not necessary for the formation of coprosterol.. It is suggested, therefore, that coprosterol is formed by the action of intestinal bacteria on the cholesterol secreted into the intestine through the intestinal wall.

Hyperinsulinism.

I. M. RABINOWITCH AND A. F. FOWLER (*Journal of Nutrition*, February, 1935) have recorded studies of the simultaneous blood sugar and respiratory metabolism time curves obtained from five cases of spontaneous hyperinsulinism. The patients complained of weakness, hunger and fatigue appearing from half to three hours after meals. The symptoms were relieved by ingestion of carbohydrate foods. After administration of glucose the blood sugar time curves were characteristically flat. In the absence of defective absorption of glucose from the gastro-intestinal tract these curves indicate an exceptionally good carbohydrate tolerance. According to the respiratory metabolism data, the response to glucose ingestion was either oxidation or storage. The type of response appeared to depend on the amount of glycogen in the body; when the glycogen was low the tendency was toward storage, and when the diet was liberal in respect to carbohydrate the tendency was toward oxidation. The symptoms, which were essentially the same in all cases, appeared to be independent of the glycogen content of the body and were due to reduction of the glucose content of the blood. The authors state that the condition resembles insulin shock due to insulin injections.

The Sedimentation Test.

THE factors influencing the rate of sedimentation of red cells have been discussed by F. Boerner and H. F. Flippin (*Journal of Laboratory and Clinical Medicine*, March, 1935). They employed the 200 millimetre Westergren tube and expressed the sedimentation rate as the number of millimetres of sedimentation at the end of one hour. Normal blood, containing between 4·5 and 5·5 million red cells per cubic millimetre, was used. The sedimentation rate was found to vary from 2 to 4 for females, and from 1 to 3 for males. In 50 cases of secondary anæmia arising from various causes (with red cell counts varying from 1·9 to 4·25 million per cubic millimetre) the sedimentation rate varied from 8 to 130. In each of these cases the sedimentation rate was lower when the test was performed on a sample of blood after the cell concentration had been adjusted to between 4·5 and 5·5 million per cubic millimetre. Of these 50 cases, 14 showed normal sedimentation rates after adjustment of the cell concentration. The differences in the sedimentation rates between the corrected and uncorrected samples was not related to the degree of anæmia, so that a mathematical correction based on the original cell count was unreliable. In another series of 35 cases it is shown that the sedimentation rate falls appreciably if the test be performed more than one hour after collection of the blood sample, the reduction in rate being proportional to the length of delay.

The Vitamin B_2 Complex.

PAUL GYÖRGY (*Biochemical Journal*, March, 1935) reports the results of studies which are directed to the elucidation of many of the difficulties in interpretation of earlier work on the vitamin B_2 complex. It was found possible to produce with regularity cessation of growth and symptoms of dermatitis (the so-called "rat-pellagra") in rats by feeding a vitamin *B* free diet supplemented with pure crystalline or highly purified vitamin B_1. The addition to this diet of lactoflavin did not prevent the onset of dermatitis and did not permit growth. When a yeast supplement (a charcoal eluate made according to the method of Peters) was added to the basal diet, there was still no growth, but the symptoms of rat-pellagra were not produced, although there appeared irregularly a skin disease which was quite distinct from the classical rat-pellagra and was considered to be due to absence of lactoflavin. When both lactoflavin and the yeast supplement were added to the basal diet, normal growth and absence of skin disease were observed. The yeast supplement is apparently identical with Chick's "Y-substance" and has been provisionally named vitamin B_6 by György, who considers that the growth-promoting function of vitamin B_2 is due to lactoflavin *plus* vitamin B_6, and that the pellagra-preventing factor is vitamin B_6 alone.

Adrenal Cortical Hormone.

A. GROLLMAN, W. M. FIROR AND E. GROLLMAN (*Journal of Biological Chemistry*, April, 1935) have described a simple method for the preparation of the adrenal cortical hormone in a form suitable for oral administration. Fresh adrenal glands from pigs were defatted and packed in carbon dioxide snow for transport. The glands were ground finely in about three times their volume of acetone. The acetone extracts contained 95% of the hormone. The extract was filtered and the filtrate was evaporated *in vacuo* to remove all the acetone. The residue was filtered, neutralized and shaken with activated charcoal, which adsorbed the hormone. After a series of washings the charcoal-hormone complex was suitable for biological tests, which were performed on adrenalectomized rats and dogs. Control animals died within a week after adrenalectomy. When the charcoal-hormone complex was given orally to adrenalectomized animals they grew normally and survived as long as an adequate amount of the hormone was given. When the charcoal-hormone was omitted from the food the animals then suffered adrenal insufficiency and died. Extracts suitable for parenteral administration could be prepared easily from the charcoal-hormone complex. It is considered that, in the gastro-intestinal tract, the hormone is liberated from the charcoal and then absorbed.

Familial Renal Glycosuria.

M. S. BROWN AND R. POLESCHUCK (*Journal of Laboratory and Clinical Medicine*, March, 1935) have described four cases of renal glycosuria occurring in one family. It is suggested that the condition was inherited. There was no evidence of *diabetes mellitus* in any of these patients. The first patient was a male, aged sixty years, who had been known to have glycosuria for seventeen years. Of his five children, two had persistent glycosuria, two gave repeatedly normal results to tests for urinary glucose, and in one glucose had been noted in the urine on one occasion, but repeated tests gave normal results. The only grandchild had persistent glycosuria. All these patients exhibited normal glucose tolerance curves.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Medical Society Hall, East Melbourne, on June 5, 1935, DR. WALTER SUMMONS, the Vice-President, in the chair.

Vaginal Discharges.

DR. HUBERT JACOBS read a paper entitled "The Investigation and Treatment of Vaginal Discharges" (see page 101).

DR. ROY W. CHAMBERS congratulated Dr. Jacobs on his manner of presenting the subject. He thought that, to an extent, doctors were to blame for the persistence of vaginal discharges. He had often heard a patient say that her doctor had told her not to worry about her discharge or to use a douche with a few useless drops of lysol added; the doctor might not have examined the patient or might have examined her in a totally inadequate fashion. Other women, with or without advice, went to the other extreme and used douches containing a strong percentage of some irritating substance which made the condition considerably worse. *Pruritus vulvæ* was a common and distressing symptom due to alterations in the skin by long-continued discharge. Owing to lack of interest and to inefficient treatment many women had discharges for years, which, being disregarded, might lead to serious results. Dr. Jacobs had given an excellent *résumé* of recent work on this subject and had left little to be added or to criticize; and Dr. Chambers was content to stress some of the more important points. For success to be attained in the treatment of this condition thorough investigation and accurate ætiological diagnosis were necessary. Dr. Jacobs had divided the common vaginal discharges into four main groups: (*a*) trichomonad, (*b*) mycotic, (*c*) infective vaginitis, such as gonococcal, (*d*) non-infective leucorrhœa of virgins and others.

To treat all these conditions in the one way and with the one agent would mean a very small percentage of improved or of cured patients. To obtain definite information as to the causative factor was usually very easy. The clinical picture was significant to the expert, but, when in doubt, one could use the microscope. Nearly as much information could be obtained by examining a drop of fresh discharge at body temperature as from a stained slide. Dr. Chambers concurred entirely with Dr. Jacobs in his strong disapproval of the haphazard use of douches in treatment. He felt certain that in lots of cases douches, apart from being of any benefit, often made matters worse. He liked a douche to be used only to clear away some of a very profuse discharge, but did not expect douches to exert any curative influence apart from the action as an irrigant. He heartily applauded Dr. Jacobs's statement that the cavity of the uterus was rarely responsible for the presence of any of the purulent or infective discharges and that, therefore, dilatation and curettage as a method of treatment for vaginal discharge was, in most cases, to be thoroughly condemned. Another point of some importance was the great need for care in the treatment of gonococcal endocervicitis in the acute and in the subacute stages. Frequently in such circumstances the cervix was swabbed out with cotton wool applicators soaked in various solutions. There was a very grave risk of pushing such an applicator into the cavity of the uterus. If this were done when the menstrual period was at hand, there was a great risk of extension of the disease to the tubes. Nature seemed to have provided some sort of barrier to the upward spread of infection about the region of the internal *os cervicis* and it was wise not to interfere with this zone.

With regard to the treatment of the various types of discharge, Dr. Jacobs had indicated the methods he had found most satisfactory. Dr. Chambers thought that universal agreement had been reached that the use of gentian violet solution was most suitable for the mycotic type. Treatment of trichomonad vaginitis was not so straightforward; there was considerable variations in the methods advocated by the different authorities. While in agreement with Dr. Jacobs that the method which had been described by him was the one most likely to produce the greatest number of successful results, Dr. Chambers was not so optimistic about the length of time taken to achieve these results. Lots of workers had claimed good results with the use of mercurochrome, picric acid and various other dyes. Another group advocated what might be called the dry method. The vagina was filled daily with some form of powder, of which sodium bicarbonate was the common example. Sodium bicarbonate and acetarsone were a favoured combination. These powders were introduced into the vagina by means of a special powder blower. Dr. Chambers had found, with Dr. Jacobs, that hexyl-resorcinol was most satisfactory, but he preferred the use of certain suppositories as a substitute for the douche. The suppository that he had found most useful was known as "Gynex Number 1" and was of the "foaming" type. It contained ichthyol and hexyl-resorcinol.

He would like to hear some discussion concerning the desirability of persisting with the treatment of trichomonad vaginitis during the menstrual period. He felt that if untreated at this time the disease appeared to undergo exacerbation. In conclusion he wished to emphasize the need for great patience and perseverance on the part of the medical attendant in order to get successful results in most cases of vaginitis. In addition, the patient was called upon also to show tremendous patience and conscientious cooperation.

DR. E. R. WHITE remarked that the condition under discussion was apt to haunt the gynæcologist. Apart from specific infections, the common leucorrhœal complaints arose from chronic cervicitis. Rarely was the body of the uterus at fault. Realizing this, he divided the patients into two main classes, according to age and for the purpose of differentiating principles of treatment. The first group consisted of the older women, approaching or past the menopause, with chronic cervicitis, thickening of the cervix, and discharge visibly coming from the cervical zone. Dr. White thought that the best treatment for this type of case was surgical treatment. Sometimes operative removal of portion of the cervix was sufficient, but in the presence of menorrhagia, when the uterus was quite likely to be in a pre-cancerous state, total hysterectomy was the operation of choice. The second group to which he wished to refer was made up of those younger women with chronic leucorrhœa associated with pains, especially sacral backache. Usually retrodeviation of the third degree was found in these cases and leucorrhœa became a daily disability. In such cases he advocated internal shortening of the round ligaments with or without surgical diathermy applications to the endocervix. He found that judicious use of electro-coagulation often relieved patients of leucorrhœa, but he believed that it was a golden rule not to indulge in surgery of the cervix during the fertile period. He used very moderate and very careful electro-coagulation to avoid local damage. He thought, too, that there was a field of usefulness here for medical diathermy, but the whole of the endocervix had to be included in the treatment. He felt that the subject of vaginitis was a bugbear to the general practitioner and to the gynæcologist alike, and he was very grateful to Dr. Jacobs for his thorough and scientific exposition of an important and difficult subject.

Dr. White mentioned that radium was used a great deal in gynæcology for menorrhagic patients approaching the menopause. By the use of radium an artificial menopause could be effected, but he would not advise this procedure for leucorrhœal vaginitis alone.

DR. B. MILNE SUTHERLAND stated that the more recent advances had been brought forward by Dr. Jacobs and he would like to join in laying stress on the essential nature of the investigation that should precede and accompany treatment in these cases of vaginitis.

He was sorry that Dr. Jacobs had not said more about the post-traumatic causes of vaginitis. These arose from traumata of child-bearing and included as examples most married women. He reminded members of the dictum of

the late Dr. Rothwell Adam, that princesses might be pregnant and queens have gonorrhœa. Trichomonad discharges were frequently to be seen in Melbourne, but monilias were seen but rarely. It was important to know when one was dealing with trichomonads. He regarded treatment in the menstrual period as essential because until the last trichomonad was destroyed it was impossible to clear the condition up, and when treatment was suspended, even for a few days, the organisms would make headway. When an examination was being made for the presence of this infection, it was necessary to have the slide warm and the discharge absolutely fresh, or only the round-celled form would be seen and not the spindle-shaped form. At other times a cause for a discharge could not be traced and only a mass of squamous-celled epithelium might be found on the slide, yet the discharge might cause a lot of worry. After cervical treatment, correction of subinvolutional malposition *et cetera*, a practitioner might be induced to do hysterectomy and, later on, the leucorrhœal discharge was just as bad as ever. It was not fair to the subject to do hysterectomy for leucorrhœa alone.

Like Dr. Chambers, Dr. Sutherland was not so optimistic as Dr. Jacobs about the length of time and the amount of treatment required for a cure. He thought it preferable to treat patients over a longer period of time to be safe. The approach to the patient, mentioned by Dr. Jacobs, should be stressed. The patient must be told that treatment might take a long time and that a great deal of patience and faithful cooperation would be required.

DR. J. R. HEATH expressed his interest in Dr. Jacobs's remarks about the efficacy of injections of theelin in gonococcal vaginitis in childhood. During the past few months he had been trying this method of treatment in a number of resistant cases, but had not realized that it would be possible to do away with local treatment entirely. When he had used theelin he had been gratified with the way in which the patients responded.

DR. SYDNEY CRAWCOUR referred briefly to a measure of success that he had had in the treatment of trichomonad vaginitis by the use of oil of titrol, but mentioned that the condition was apt to recur.

Dr. Jacobs, in reply, expressed his pleasure at the way in which his paper had been received and at the turn the discussion had followed. He did not mean to be dogmatic when he recommended hexyl-resorcinol in the treatment of trichomonad vaginitis. He realized that there were other preparations that could be used with success, but he favoured hexyl-resorcinol because it was economical, non-irritating and did not cause a stain. From his reading and his experience he felt justified in stating that trichomonads had never been found to be present in the cervix, although he had referred previously to Kleegman's "strawberry cervix". It was not necessary, therefore, to put any antiseptic into the cervix. He would like to emphasize the importance of getting the discharge out of the posterior fornix. He felt that this area was particularly favoured by the trichomonads and was most likely to be missed when the "S.T.37" was applied, because it was covered by the speculum. The "S.T.37" was bland and non-irritating to the vaginal mucosa and, when applied after thorough removal of the discharge, fixed a film over the mucosal cells. In spite of what had been said, he still thought that about twelve treatments was a fair average number in which to expect a cure. Perhaps the conditions under which he worked in specially fitted rooms would give him a big advantage over the average general practitioner and perhaps his thorough treatment of the posterior fornix made a material difference to the number of treatments before cure followed and to the freedom from recurrence in a shorter time. He had an objection to the reliance on pessoids as the mode of application of the antiseptic used because the antiseptic would not have much chance of getting into the posterior fornix.

He had satisfied himself that mercurochrome in 20% solution settled trichomonads, but he had not published his results because of the cost of mercurochrome and the success of "S.T.37". He was always careful not to encroach on to the internal os when he used an applicator.

Dr. Jacobs had purposely not stressed the vaginal discharges arising from traumatic infections, because of the limits of time and space, but he considered that it was dramatic how pain and menorrhagia disappeared after linear cauterization. He used a fine nasal type of tip on the electric galvano-cautery, and by delicate light applications could trim up the cervix to look like a normal virginal cervix.

After radium application there was a tendency for the formation of the senile type of filmy adhesions in the vaginal vault, and it was important that these should be broken down. He thought that the thin watery discharge from the uterus after radium treatment was due to the dissolution of radium membrane which had developed in the endometrium.

In regard to the female sex hormone, Dr. Jacobs described some interesting features of a patient who had a vaginal discharge regularly on the seventeenth day of the sexual cycle. On examination he discovered a white raised patch suggestive of thrush, without any surrounding zone of hyperæmia. This patch consisted of an accumulation of epithelial cells. He was convinced that in this case the discharge was due to excess of œstrin. It was difficult to know how to reduce the œstrin when it was in excess, other than by producing artificial menopause by the application of radium.

He referred Dr. Heath and others interested in the theelin treatment of vaginal discharges to the *American Journal of Obstetrics and Gynecology* of April, 1935, and especially to the summary on page 558. He thought that relapses were often due to reinfection from the urethra, the pavement transitional type of epithelium of which would not be influenced by the theelin and would require local treatment. It was of interest to note Miller's conclusion that acute recent infections required larger and longer treatment than was needed for chronic cases.

In conclusion, Dr. Jacobs thought that further use should be made of the information that was available regarding the significance of the degree of acidity of a vaginal discharge. He mentioned that the hydrogen ion concentration could be estimated quickly and sufficiently accurately by means of the simple capillator put up by British Drug Houses.

ANNUAL MEETING, MELBOURNE, 1935.

Provisional Programme.

THE following provisional programme has been issued by the Executive Committee of the annual meeting of the British Medical Association to be held in Melbourne in September.

Monday, September 9, 1935.

Arrival and disposal of interstate and overseas visitors.

9 a.m. to 6 p.m.—Ladies' club and registration rooms (reception rooms) open at Oriental Hotel (tickets for excursions available).

9 a.m. to 5 p.m.—Registration rooms at University of Melbourne open. Tickets for Monday, Tuesday and Wednesday (excursions), tickets for annual dinner, tickets for National Temperance League's annual breakfast, tickets for Irish Medical Schools and Graduates' Association luncheon, tickets for medical missionary breakfast.

2.15 p.m.—Official opening of trade exhibition and hobbies exhibition.

2.30 p.m.—Excursions.

4 p.m.—Civic reception in Town Hall by the Lord Mayor to officials, official delegates and certain section officers. Afternoon teas.

5 p.m.—Late afternoon parties.

7 p.m.—Dinner by Victorian Medical Women's Society to overseas and interstate medical women at the Wattle Café, Little Collins Street.

9 p.m.—Reception by State Government at National Gallery. A number of small dances.

Tuesday, September 10, 1935.

9 a.m. to 5 p.m.—Registration rooms open. (All tickets available, except excursions for Thursday, Friday and Saturday.) Ladies' club open. Trade exhibition open.
9 a.m.—Conference between officials of Central Council and the Federal Council, adjourned from London.
10 a.m.—Ladies' excursions. Visits to National Gallery and Shrine of Remembrance, followed by morning tea. Whole-day excursions.
10 a.m.—Pathological museum open. Hobbies exhibition open.
11 a.m.[1]—Adjourned annual general meeting and presidential address at Town Hall, Melbourne. Members and ladies will be seated by 10.45 a.m.
1 p.m.—Luncheon parties. Section officers' luncheon.
2.30 p.m.—Half-day excursions.
3.30 p.m.—Afternoon teas.
4 p.m.—Members will robe in Chapter House for the official religious service.
4.30 p.m.[1]—Official religious service at Saint Paul's Cathedral.
5.30 p.m.—Late afternoon parties.
7 p.m.—Ladies' official dinner. Dinner parties. Returned medical officers' dinner.
8.30 p.m.[1]—Presidential reception at Town Hall, Melbourne.

Wednesday, September 11, 1935.

8.45 a.m.—Clinical address.
9 a.m. to 5 p.m.—Registration rooms open. (Excursion tickets for Thursday, Friday and Saturday available.) Ladies' club open. Trade exhibition open. Hobbies exhibition open. Whole-day excursions.
10 a.m. to 1 p.m.—Scientific section meetings. Ladies' excursions. Visits to places of interest—hospitals, shops, factories, schools, art galleries *et cetera*.
10 a.m.—Junior American tennis tournament at Kooyong (all day).
1 p.m.—Irish Medical Schools and Graduates' Association luncheon. Rotary luncheon.
2 p.m.—Half-day excursions.
3 p.m.—Bridge party by twelve hostesses at Number 9, Darling Street. Conferring of honorary degrees by the University of Melbourne, followed by a garden party.[1]
5.30 p.m.—Late afternoon parties.
7 p.m.—Dinner parties.
9 p.m.—Reception by His Excellency the Governor and Lady Huntingfield at Government House.

Thursday, September 12, 1935.

8.30 a.m.—National Temperance League annual breakfast.
8.45 a.m.—Clinical address.
9 a.m. to 5 p.m.—Registration rooms open. Ladies' club open. Trade exhibition open. Pathological museum open. Hobbies exhibition open. Whole-day excursions.
9.30 a.m.—Junior golf tournament (all day).
10 a.m. to 1 p.m.—Scientific sections. Excursions, with luncheon at Mount Eliza and tea at Frankston (several hostesses).
1 p.m.—Luncheon parties.
2 p.m.—Half-day excursions. Tennis at Kooyong. Bridge.
5.30 p.m.—Late afternoon parties.
7.15 p.m.—Annual dinner at Myer's.
9.30 p.m.—Ball given by members of the Victorian Branch at Earl's Court, St. Kilda.

Friday, September 13, 1935.

8.30 a.m.—Medical missionary breakfast at the Victoria Palace.
8.45 a.m.—Clinical address.
9 a.m. to 5 p.m.—Registration rooms open. Ladies' club open. Trade exhibition open. Pathological museum open. Hobbies exhibition open.
9 a.m.—Whole-day excursions. Ladies' excursions: Macedon, with luncheon; Healesville, with luncheon. Golf: Leinster and Childe Cups for men at Metropolitan Club; Notts Ladies' Challenge Cup at Peninsula Club, Frankston. Luncheon and tea.
10 a.m. to 1 p.m.—Scientific sections.
1 p.m.—Luncheon parties.
2 p.m.—Half-day excursions.
2.30 p.m.—Conferring of honorary fellowships by Royal Australasian College of Surgeons.
5.30 p.m.—Late afternoon parties.
7 p.m.—Dinner parties.
8 p.m.—Popular lecture by the Right Honourable Lord Horder. Theatre party.
9 p.m.—Army, Navy and Air Force Services' Ball (by invitation).

Saturday, September 14, 1935.

9 a.m.—Excursions (half-day, day and week-end). Melbourne Medical Association excursion and picnic luncheon. Ladies' excursions (Flinders and luncheon; Portsea and luncheon, with tea at Dromana; Acheron Way and luncheon). Excursion to Korumburra, inspection of Bush Nursing Hospital, luncheon and return via Pakenham, and tea at Bush Nursing Hospital, or return via Yallourn.
1 p.m.—Luncheon parties.
2 p.m.—Moonee Valley races.
2.45 p.m.—Football at Melbourne Cricket Ground for overseas visitors. Tea at Royal Aero Club at Essendon Aerodrome.
5.30 p.m.—Late afternoon parties.

[1] Academic dress.

Notification of Intention to Attend the Annual Meeting.

As has been stated in the journal of July 13, it is of the utmost importance to the success of the meeting that early notification of intention to be present be made at an early date to the Honorary Local General Secretary, British Medical Association Office, 12, Collins Street, Melbourne.

In addition to the need for this for general purposes, it is particularly necessary for at least two functions—the reception given by His Excellency the Governor and Lady Huntingfield at Government House, and the State Government reception at the National Gallery. The invitations to these functions will be sent direct from the Governor or State Government to members at their own addresses on receipt of a list of names from Dr. Major, and those desirous of receiving such invitations must notify Dr. Major at the above address not later than August 15. The member should state also if he will be accompanied by a lady or not.

Academic Dress.

Academic dress will be worn at the official religious service to be held at Saint Paul's Cathedral at 4.30 p.m. on Tuesday, September 10; on the occasion of the adjourned annual general meeting and President's address on Tuesday morning in the Town Hall, Melbourne, and at the President's reception on Tuesday evening; also at the conferring of honorary degrees and the garden party at the University on Wednesday, September 11.

The Annual Dinner.

The annual dinner will be held in the mural hall and grill room at Myer's on Thursday evening, September 12. The price of the tickets will be £1 1s. (inclusive of wines). These rooms intercommunicate, one seating 500 and the other 300, and loud speakers are already installed so that the speeches will be heard equally well in either room. Apart from provision for the official guests, the allotment of seats at this dinner will be made early in the week of the meeting, by ballot if necessary. Apart from certain official lady guests, it is impossible to allot tickets to any non-medical women until it is known how many members desire to be present.

Those desirous of attending are requested to make early application to the Honorary Secretary, Dinners Subcommittee, British Medical Association Office, 12, Collins Street, Melbourne.

Clinical Addresses and Hospital Demonstrations.

In addition to the scientific section meetings, the following special clinical addresses have been arranged. They will be of such a nature as to appeal to the general practitioner as well as the consultant.

"Specialism: Neurology and Medicine", by Professor Edwin Bramwell (Edinburgh).
"The General Practitioner and Forceps", by Sir Ewen Maclean (Wales).
"The Valley of Dry Bones", by Professor E. W. Hey Groves (Bristol).
"The Dyspepsias of Childhood", by Dr. Robert Hutchison (London).
"Œsophageal Obstruction", by Mr. H. S. Souttar (London).

A subcommittee has also arranged a comprehensive programme of demonstrations at the following hospitals: Royal Melbourne, Alfred, Saint Vincent's, Women's, Children's, Eye and Ear, the Orthopædic at Frankston, and at Gresswell Sanatorium.

Hotel and Boarding House Accommodation.

Below will be found a list of hotels and boarding houses in Melbourne recommended for the accommodation of visitors to the annual meeting. Application should be made direct to the hotels and boarding houses, mentioning the British Medical Association.

The charges stated may be subject to slight alteration. All the hotels of Melbourne are approximately three-quarters to one mile from the University. There are a number of small unlicensed guest houses situated in the environs of Melbourne, details of which are as yet unavailable.

Excursions and Tours.

For interstate visitors attending the meeting special motor trips are being arranged from Brisbane and Sydney via Prince's Highway, including Canberra, if desired, both to and from Melbourne. Also from Sydney a special railway tour will be available on Thursday, September 5, and Friday, September 6, via Canberra, Hume Weir, Kiewa Valley, Tawonga Gap and Mount Buffalo, arriving in Melbourne at noon on Sunday or Monday, according to the day of departure.

From Adelaide similar tours will leave on Wednesday, September 4, and Thursday, September 5, including two days at the Grampians, and arriving in Melbourne at 9.30 p.m. on Saturday or Sunday, according to the day of departure.

All these tours will be available after the meeting in the reverse way for visitors returning home. Full details will be supplied by the Excursions Officer.

After the meeting seven extended tours by motor have been arranged to various parts of Victoria of two to four days' duration, while a railway week-end tour to Mount Buffalo leaves on Friday, September 13, at 4 p.m. Also a camping and fishing week-end may be taken at the Metropolitan Board of Works quarters at Wallaby Creek, at the Plenty Ranges watershed.

At midnight on Sunday, September 15, a railway Reso tour of Victoria commences. This will embrace the western district, visiting station homesteads at Camperdown, the Trufood factory at Terang, whilst at Warrnambool a kangaroo drive will take place. Albury and Hume Weir are next visited and then the wineries at Rutherglen. A day at the Chalet, Mount Buffalo, follows, then Bright, with its plantations, and Melbourne is reached on Friday evening.

Travelling Facilities.

The Commissioners of Railways in the different States have granted the following concessions for members attending the meeting: (i) Return tickets from other States to Melbourne at single fare, plus one-third, for interstate members. (ii) Holiday excursion fares for members travelling from Victorian country stations to Melbourne.

These tickets are available for one month and any member may obtain a similar concession for wife or husband and for children (sons under the age of sixteen years or daughters of any age who are living with and dependent upon their parents for support), provided they accompany the member and separate certificates are furnished.

These concessions will be available only to members who present the prescribed certificate signed by the Secretary of the Branch in the State from which members travel.

The Union Steamship Company of New Zealand and Huddart, Parker, Limited, have consented to allow a concession of 10% off the first class fares of members and their

Name and Address.	Total Accommodation.	Charges.			
		Bed and Breakfast. (Per Day.)	Per Day.	Lunch.	Dinner.
MELBOURNE.					
Menzies Hotel	220	16/–	27/–	5/–	8/6
Hotel Alexander	350	15/–	£5/5/– wk.	3/6	6/6
Hotel Windsor	300	12/6 16/6	21/– 25/–	4/–	6/6
Oriental Hotel[1]	120	10/6	20/–	4/–	7/6
Scott's Hotel	180	11/6	21/6	*à la carte*	
Federal Hotel	500	7/– 12/–	15/– 21/–	3/6	5/–
Victoria Palace (unlicensed)	850	5/– 10/–	8/– 17/6	*à la carte*	
New Treasury Hotel	45	8/6	15/–		
St. Ann's, 2, Commercial Road (off St. Kilda Road)	4 single, £3/3/– per week, inclusive 4 double, £6/6/– per week, inclusive.				
ST. KILDA.					
George Hotel	125	9/– 13/6	£5/5/– wk.	4/6	7/–
Esplanade Hotel	120	9/–	20/–	3/–	4/–
Chevron (unlicensed)	200	10/6	15/–	3/–	4/–

[1] It is understood that accommodation at the Oriental Hotel is fully booked.

wives travelling from New Zealand to Melbourne and return. Tasmanian Steamers Proprietary, Limited, offer the same concessions to those travelling from Launceston, Burnie, or Devonport, as do The Adelaide Steamship Company, Limited, Australian Steamships Proprietary, Limited, Australasian United Steam Navigation Company, Limited, Huddart Parker, Limited, McIlwraith, McEacharn, Limited, and Melbourne Steamship Company, Limited, for those members who come from Western Australia, South Australia, Queensland and New South Wales.

To obtain these shipping concessions it will be necessary to produce a certificate from the Secretary of the local Branch certifying that they are *bona fide* members attending the meeting.

General Information for Ladies.

The ladies' club will be at the Oriental Hotel, Collins Street, and a cordial invitation will be extended to all lady members of the Association, and to ladies accompanying members attending the meeting to make use of the club.

All ladies will be requested to register their names at the club on arrival, where badges will be issued to them, together with a copy of the "Handbook for Ladies".

The club will be open from 9 a.m. to 6 p.m., and will contain a lounge, writing and card rooms, also a hairdressing saloon will be available. Morning tea, luncheon, afternoon tea will be served as required. Letters, telegrams, messages *et cetera* may be directed to the club, at which there will also be a general information bureau.

All information regarding the ladies' programme will be obtained at the club, as well as information of excursions, golf, tennis *et cetera*.

A Junior Ladies' Committee is arranging a programme of similar entertainments for the younger visitors.

Correspondence.

VACCINATION AGAINST SMALLPOX.

SIR: The note in the journal (July 6, 1935, page 22) with reference to the work of Pinto at Bombay on the duration and degree of immunity conferred by vaccination raises some interesting questions of practical importance. It was Force, of San Francisco, who in 1913 suggested the practical use of the "immunity reaction" to vaccination which Pirquet had noted seven years before. Since that time this immunity reaction has been widely used as a routine administrative criterion in quarantine practice, and for some years it has been adopted in the Australian Quarantine Regulations and standing instructions for quarantine officers of the Commonwealth Department of Health.

The suggestion of your reviewer that an investigation along similar lines in Australia would give useful information is already met to some degree by the observations of officers of the Commonwealth Department of Health on which the relevant quarantine practice is based. The decline in vaccination of infants and children in Australia would probably make any local investigations on the lines of Pinto's work difficult, if not impracticable. Some comparable records are, however, available for persons of all ages in a note on "The Criterion of Immunity in Vaccinated Persons", by Dr. F. McCallum in *Health* for January, 1927, page 20. Using groups practically identical with those adopted by Pinto, this note gives a table which clearly shows the gradual decline of immunity amongst 939 persons revaccinated on board a vessel recently quarantined in Australia. Accelerated reactions were classified according to whether maximum reaction and areola subsided by the fifth day (++) or by the eighth day (+). Including for comparison results of vaccination amongst 133 persons on the vessel never previously vaccinated, the table showed the results set out below.

This note also included records showing the relation of size and character of scars of previous vaccinations to the results of revaccination. The following conclusions were recorded:

1. Average size of scars does not appear to diminish appreciably over a period of years.
2. Character of scars appears to become less marked over a period of years.
3. Immunity as gauged by reaction to revaccination appears to bear no definite relation to size or character of scars of previous vaccination.
4. The factor of importance in determining immunity is the time that has elapsed since previous vaccination.
5. It cannot be definitely stated that immunity is absolute in every individual over a prescribed period of time, although probably the arbitrarily accepted period of seven years is the average duration of a relatively high degree of immunity.
6. Revaccination, interpreted in terms of the classification of results stated, provides a practical method of assessing the degree of immunity in any individual.

These vaccinations and observations were carried out according to the standard technique adopted by quarantine officers of the Commonwealth Department of Health. The results correspond with Pinto's figures and with recorded observations made in many countries, especially in the United States of America.

It might be noted that the International Sanitary Convention for Aerial Navigation (1933), which prescribes the quarantine code for international air traffic and to which Australia has subscribed, defines persons who shall be considered immune to smallpox as those (*a*) who can produce proof of a previous attack of smallpox, or if they have been vaccinated within less than three years and more than twelve days, or (*b*) if they show local signs of early reaction attesting an adequate immunity.

It will thus be seen that the recent observations of Pinto confirm administrative practice and Australian quarantine procedure which have existed over some years.

Yours, etc.,

J. H. L. CUMPSTON,
Director-General of Health.

Department of Health,
Canberra,
July 9, 1935.

Reaction Result of Vaccination.	Previously Vaccinated or Revaccinated.								Never Previously Vaccinated.	
	Within 7 Years.		Within 7-15 Years.		Within 15-30 Years.		More than 30 Years Ago.			
	No.	%	No.	%	No.	%	No.	%	No.	%
Immunity	78	42·9	21	11·2	9	2·5	4	2·0	..	..
Accelerated ++	52	28·6	52	27·7	30	8·2	2	1·0	2	1·5
Accelerated +	47	25·8	100	53·2	224	61·0	81	40·1	14	10·5
Typical Jennerian	5	2·7	14	7·4	97	26·4	115	56·9	111	83·5
Negative on three occasions	..	..	1	0·5	7	1·9	..	..	6	4·5
Total	182	100·0	188	100·0	367	100·0	202	100·0	133	100·0

THE "CHEST" PHYSICIAN AND THE RADIOLOGIST.

SIR: Two fundamentally wrong opinions are contained in the reply of ". . . Angels Fear to Tread" in the journal of the 15th instant.

May I have a final opportunity to help your correspondent in his difficulties?

1. The radiologist must be regarded as a medical consultant. His opinion concerns the patient and is not merely an opinion on an X ray plate. This involves the interpretation of the radiographic findings and their application concerning which is the second error.

2. The radiologist as interpreter of the X ray plate would give the correct "translation" of the report being considered somewhat as follows: There is relative (or absolute) loss of radiolucency of the lower third of the right lung field reaching to the axilla laterally. The rest of the right lung and the whole of the left are radiographically normal. Usually the character of the upper margin of the dulness, its position from front to back and the position of the heart would be stated.

The radiologist applying his knowledge of correlated pathology would know that such radiographic alterations could in certain instances be due to generalized or encysted pleural effusion, pneumonia of various types, pleural or pulmonary hæmorrhage, atelectasis, actinomycosis, new-growth *et cetera*.

The radiologist applying his experience and clinical sense can be a powerful ally with any "chest" physician in determining the disease present in any particular patient as most likely to account for the radiographic findings. If the radiologist is not worthy of this trust, change the radiologist, and if the physician cannot appreciate the importance of this use of radiology return him to the "halls of learning" and have him listen again to the portentous whisperings therein.

Yours, etc.,

30, Princes Street, Auckland, New Zealand, June 27, 1935.

F. J. GWYNNE.

THE MORTALITY OF APPENDICITIS.

SIR: I wish to thank Dr. J. Colvin Storey for his reply to my letter.

I am sorry that I cannot supply the records of the total number of anæsthetics given by me, but an investigation into the last two or three years shows the number between three and four per week; say seven every two weeks. This estimate is on the conservative side, as during the last two or three years there has been greater use of local anæsthesia than formerly. This would make the total general anæsthetics about 182 *per annum*, or roughly two thousand during my residence in Bingara. This, of course, includes all anæsthetics for major and minor operations, dental anæsthetics, and those given for a colleague in a neighbouring town.

I therefore apologize to you, sir, your readers, and Dr. Storey for the use of so loose a word as "several" thousand, when "two" was the correct word.

With very few exceptions all these have had the ethyl chloride-ether sequence or ethyl chloride alone, and I have not seen the dangerous collapse phenomena as stated by Dr. Storey and those taking part in the discussion on the subject (THE MEDICAL JOURNAL OF AUSTRALIA, July 6, 1935).

I still maintain that the statement that the ethyl chloride-ether sequence is dangerous and should be forbidden will come as a great surprise to most general practitioners.

Yours, etc.,

Bingara, New South Wales, July 8, 1935.

S. SHINEBERG.

SYMPATHECTOMY IN RETINITIS PIGMENTOSA.

SIR: Dr. Temple Smith's quotation from *The British Journal of Ophthalmology* misses the point, for I never do a cervical sympathectomy for *retinitis pigmentosa*. The operation carried out by me is a superior thoracic ganglionectomy, which gets all the sympathetic fibres going by the carotid arteries and the vertebral artery to the brain. Cervical sympathectomy, the English variation, does not do this. In addition, one of my earliest patients underwent operation over five years ago and has now full fields and $^6/_6$ central vision. There are other patients who have shown satisfactory improvement also, but the period over which the improvement has taken place is not as great as in the case of the first patient.

Yours, etc.,

185, Macquarie Street, Sydney, July 9, 1935.

N. D. ROYLE.

CHRONIC INFECTIVE ARTHRITIS.

SIR: Referring to the report in the journal of July 6 on the series of cases of this disease treated by Crowe's vaccine, and some of which were shown by me at a clinical meeting at the Royal Melbourne Hospital recently, I would draw your attention to an error in that report.

In each instance the vaccine used in treatment was mixed "M.D." and "Streps.", and not "M.D." alone. As I have had some inquiries since that report appeared, I should be obliged if you would insert this note in your journal.

Yours, etc.,

61, Collins Street, Melbourne, C.1, July 16, 1935.

REGINALD HOWDEN.

Corrigendum.

IN the paper on gastric ulcer from the surgical point of view, by Dr. Howard Bullock, published in the issue of July 13, 1935, two errors have occurred. On page 46 the nineteenth line in the first column should read: "Einhorn and Crohn discuss 100 cases of peptic . . . ." The date in reference (9) on page 52 should be January 8, 1922, and not 1932.

Books Received.

HALFWAY TO THE HEREAFTER: AN ENQUIRY INTO THE MOTIVATION OF ATTEMPTED SUICIDE, by E. T. Stoneman, B.A., A.M., Ph.D.; 1935. Perth: T. F. Christie, Limited. Demy 8vo., pp. 96.

HEALTHY WEDDED LIFE: A MEDICAL GUIDE FOR WIVES, by G. T. Wrench, M.D., B.S.; Fourth Edition; 1935. London: J. and A. Churchill. Crown 8vo., pp. 349. Price: 6s. net.

THE DIABETIC ABC: A PRACTICAL BOOK FOR PATIENTS AND NURSES, by R. D. Lawrence, M.A., M.D., F.R.C.P.; Third Edition; 1935. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 67. Price: 3s. 6d. net.

CHRISTINE MURRELL, M.D.: HER LIFE AND HER WORK, by C. St. John, with preface by Lady Barrett, C.H., C.B.E., M.D., M.S.; 1935. London: Williams and Norgate, Limited. Demy 8vo., pp. 152. Price: 5s. net.

ROMANY REMEDIES AND RECIPES, by Gipsy Petulengro; 1935. London: Methuen and Company, Limited. Crown 8vo., pp. 58. Price: 2s. net.

POCKET MONOGRAPHS ON PRACTICAL MEDICINE: INSOMNIA AND OTHER DISTURBANCES OF SLEEP, by E. Miller, M.A., M.R.C.S., L.R.C.P., D.P.M.; 1935. London: John Bale, Sons and Danielsson, Limited. Foolscap 8vo., pp. 88. Price: 2s. 6d. net.

Diary for the Month.

AUG. 1.—South Australian Branch, B.M.A.: Council.
AUG. 2.—Queensland Branch, B.M.A.: Branch.
AUG. 5.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 6.—Tasmanian Branch, B.M.A.: Council.
AUG. 7.—Victorian Branch, B.M.A.: Branch.
AUG. 7.—Western Australian Branch, B.M.A.: Council.
AUG. 9.—Queensland Branch, B.M.A.: Council.
AUG. 13.—Tasmanian Branch, B.M.A.: Branch.
AUG. 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 20.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 20.—Tasmanian Branch, B.M.A.: Council.
AUG. 21.—Western Australian Branch, B.M.A.: Branch.
AUG. 21.—Victorian Branch, B.M.A.: Clinical Meeting.
AUG. 22.—New South Wales Branch, B.M.A.: Clinical Meeting.
AUG. 23.—Queensland Branch, B.M.A.: Council.
AUG. 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 28.—Victorian Branch, B.M.A.: Council.
AUG. 29.—South Australian Branch, B.M.A.: Branch.
AUG. 29.—New South Wales Branch, B.M.A.: Branch.

Medical Appointments.

Dr. T. C. James (B.M.A.) has been appointed Assistant Medical Officer at the Mental Hospital, New Norfolk, Tasmania.

* * *

Dr. D. M. Stewart has been appointed a Resident Medical Officer at the Adelaide Hospital, South Australia.

* * *

Dr. R. G. C. de Crespigny (B.M.A.) has been appointed Honorary Pathologist at the Mareeba Babies' Hospital, South Australia.

* * *

Dr. B. J. Lawrence has been appointed a Resident Medical Officer at the Mareeba Babies' Hospital, South Australia.

* * *

Dr. O. Barton has been appointed Government Medical Officer at Scone, New South Wales.

* * *

Dr. E. H. Derrick (B.M.A.) has been temporarily appointed Director of the Laboratory Section, Department of Public Health, Brisbane, Queensland, pursuant to the provisions of *The Public Service Acts*, 1922 *to* 1924, and *The Health Acts*, 1900 *to* 1934.

* * *

Dr. S. G. Taylor has been appointed Medical Officer of Health by the Longford Municipal Council, Tasmania.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi-xviii.

DIRECTOR-GENERAL OF HEALTH, SYDNEY, NEW SOUTH WALES: Honorary Ophthalmic Surgeon.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer.

METROPOLITAN INFECTIOUS DISEASES HOSPITAL BOARD, ADELAIDE, SOUTH AUSTRALIA: Assistant Medical Officer.

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.

PRINCE HENRY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Radiologist, Junior Resident Medical Officers.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Officers.

THE TOWNSVILLE HOSPITALS' BOARD, TOWNSVILLE, QUEENSLAND: Resident Medical Officer.

THE UNIVERSITY OF MELBOURNE, VICTORIA: Part-Time Independent Lecturer in Surgical Anatomy.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chiliagoe Hospital. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY HOSPITAL, are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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